12556

Sam.l Lewis M.D.

TRANSACTIONS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

VOLUME XIV.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM SEPTEMBER, 1887, TO JULY, 1889.

EDITED BY

W. E. HUGHES, M.D.,

MEDICAL REGISTRAR OF THE UNIVERSITY HOSPITAL; PHYSICIAN TO THE OUT-PATIENT DEPARTMENT OF THE CHILDREN'S HOSPITAL; RECORDER OF THE SOCIETY.

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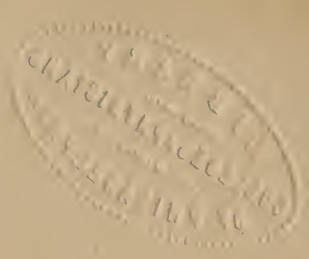

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FORMER PRESIDENTS.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. Oxon., LL.D. Cantab., elected 1857.

RENÉ LA ROCHE, M.D., elected 1858.

ALFRED STILLÉ, M.D., elected 1859, '61, and '62.

EDWARD HARTSHORNE, M.D., elected 1860 and '63.

J. M. DA COSTA, M.D., elected 1864, '65, and '66.

JOHN H. PACKARD, M.D., elected 1867 and '68.

S. WEIR MITCHELL, M.D., elected 1869.

JOHN ASHHURST, Jr., M.D., elected 1870.

JAMES H. HUTCHINSON, M.D., elected 1871 and '72.

WILLIAM PEPPER, M.D., elected 1873.

H. LENNOX HODGE, M.D., elected 1876.

S. W. GROSS, M.D., elected 1879.

JAMES TYSON, M.D., elected 1882 and '83.

E. O. SHAKESPEARE, M.D., elected 1884.

J. C. WILSON, M.D., elected 1885 and '86.

F. P. HENRY, M.D., elected 1887 and '88.

OFFICERS AND COMMITTEES

OF THE

PATHOLOGICAL SOCIETY OF PHILADELPHIA.

(Elected at the annual meeting, October, 1887.)

President.

F. P. HENRY, M.D.

Vice-Presidents.

J. H. C. SIMES, M.D.,
J. M. BARTON, M.D.,
H. F. FORMAD, M.D.,
WM. OSLER, M.D.

Secretary.

J. K. MITCHELL, M.D.

Recorder.

W. E. HUGHES, M.D.

Treasurer.

M. S. FRENCH, M.D.

Curator.

H. F. FORMAD, M.D.

Committee on Publication.

H. R. WHARTON, M.D., C. B. NANCREDE, M.D.,
J. C. WILSON, M.D., JAMES TYSON, M.D.

Committee on Morbid Growths.

WM. OSLER, M.D., GEORGE DOCK, M.D.,
G. E. De SCHWEINITZ, M.D., J. P. C. GRIFFITH, M.D.,
G. G. DAVIS, M.D., W. E. HUGHES, M.D.,
MORRIS LONGSTRETH, M.D.

Business Committee.

J. H. MUSSER, M.D., HENRY BEATES, Jr., M.D.,
W. A. EDWARDS, M.D., D. J. M. MILLER, M.D.,
JAMES TYSON, M.D.

LIST OF MEMBERS.

Members marked O. M. are those originating the Society.
Members marked N. R. are non-resident.

ELECTED

1890 Adams, J. Howe, 252 South Seventeenth Street.
1857 Agnew, D. Hayes, Emeritus Professor of Surgery in the University of Pennsylvania, 1601 Walnut Street. (O. M.)
1871 Alison, Robert H., resigned 1884.
1864 Allen, Harrison, resigned December 11, 1879.
1869 Allis, Oscar H., resigned February 24, 1881.
1887 Allyn, Herman B., 737 North Forty-first Street.
1865 Andrews, Thomas H., forfeited membership by non-payment of dues.
1871 Ashbridge, William, resigned December 11, 1879.
1867 Ashhurst, Francis, Mount Holly, New Jersey. (N. R.)
1861 Ashhurst, John, Jr., Professor of Surgery in the University of Pennsylvania, 2000 Delancey Place.
1863 Ashhurst, Samuel, resigned December 22, 1887.
1891 Ashton, Thomas G., 222 South Eighth Street.
1871 Atlee, Washington L., Jr., forfeited membership by non-payment of dues, December 10, 1874.

1866 Bache, T. Hewson, resigned December 27, 1883.
1878 Baer, B. F., resigned.
1887 Baker, G. F., 1818 Spruce Street.
1888 Baldy, J. M., 327 South Seventeenth Street.
1888 Balliet, Tilghman M., 3711 Powelton Avenue.
1876 Barr, J. W., forfeited membership by non-payment of dues, December 8, 1881.
1880 Bartholow, Roberts, 1525 Locust Street.
1868 Bartles, Wm. H., Insane Department Pennsylvania Hospital, West Philadelphia. (N. R.)
1883 Barton, Isaac, 128 South Fifteenth Street.
1869 Barton, J. M., Surgeon to the German and Jefferson Medical College Hospitals, 1344 Spruce Street.

ELECTED

1885 Beates, Henry, Jr., 1421 Girard Avenue.
1873 Beecher, A. C. W., 523 South Ninth Street.
1870 Bell, J. R. F., forfeited membership by non-payment of dues, December 10, 1874.
1871 Bennett, Wm. H., 332 South Fifteenth Street.
1863 Benton, C. H. (N. R.)
1887 Berens, Bernard, 2006 Arch Street.
1875 Bernardy, Eugene P., resigned September 22, 1881.
1870 Bertholet, Robert Morris, died 1882.
1871 Betts, Thomas, Branchtown, Pa. (N. R.)
1890 Birny, H. H., 914 Belmont Avenue.
1860 Bishop, C. S., died.
1866 Black, J. J., New Castle, Delaware. (N. R.)
1863 Boardman, C. H., St. Paul, Minnesota. (N. R.)
1886 Bodamer, George A., 1219 North Fifteenth Street.
1880 Bœnning, Henry C., forfeited membership by non-payment of dues, April 14, 1882.
1857 Boker, Charles S., Surgeon to St. Joseph's Hospital, 212 South Fifteenth Street. (O. M.)
1868 Bolles, Lucius S., died August 15, 1873.
1883 Boyd, George M., 1700 North Sixteenth Street.
1877 Bradford, T. Hewson, resigned December 23, 1880.
1873 Bray, Daniel, forfeited membership by non-payment of dues, December 8, 1881.
1883 Breinig, P. B., Bethlehem, Pennsylvania.
1890 Bricker, William H., 227 North Twenty-second Street.
1857 Brinton, John H., Professor of the Practice of Surgery and Clinical Surgery in the Jefferson Medical College, 1423 Spruce Street. (O. M.)
1891 Brooke, Benjamin. (N. R.)
1890 Brown, James M., 4023 Spring Garden Street.
1874 Brubaker, A. P., 1210 Race Street.
1874 Bruen, E. T., died March 31, 1889.
1871 Buck, William Penn, resigned October 23, 1873.
1867 Burnett, Charles H., resigned December 8, 1881.
1887 Burr, Charles W., 1330 Spruce Street.

1882 Cathcart, James H., died March 5, 1874.
1879 Cathcart, T. H., died March 28, 1882.
1887 Cattell, H. W., 222 South Thirty-ninth Street.

ELECTED

1890 Cerna, David, University of Pennsylvania.
1887 Chambers, G. H., 739 North Seventeenth Street.
1871 Cheston, C. Morris, West River, Maryland. (N. R.)
1865 Cheston, D. Murray, resigned December 13, 1883.
1887 Chrystie, Walter, 125 South Sixteenth Street.
1871 Clark, Leonardo S., resigned November 29, 1883.
1866 Cleemann, Richard A., Physician to St. Mary's Hospital, 2135 Spruce Street.
1872 Cohen, J. Solis, Professor of Diseases of the Throat and Chest in the Philadelphia Polyclinic and School for Graduates, 1431 Walnut Street.
1883 Cohen, S. Solis, Physician to the Philadelphia Hospital, 219 South Seventeenth Street.
1887 Collins, James, 704 Franklin Street.
1890 Conrad, J. Reed, 109 South Twenty-second Street.
1890 Coplin, W. M. L., 310 Catharine Street.
1869 Corbit, William B., Washington, D. C. (N. R.)
1877 Cruice, R. B., resigned December 22, 1887.
1871 Curtin, Roland G., Physician to the Philadelphia Hospital, and to the Presbyterian Hospital, 22 South Eighteenth Street.

1857 Da Costa, J. M., Professor of the Theory and Practice of Medicine in the Jefferson Medical College, 1700 Walnut Street. (O. M.)
1885 Daland, Judson, 319 South Eighteenth Street.
1859 Darby, J. T., died.
1876 Darlington, W. L. (N. R.)
1857 Darrach, James, 5094 Green Street, Germantown. (O. M.)
1868 Darrach, William, died January 28, 1881.
1887 Davis, E. P., resigned December 5, 1888.
1882 Davis, G. G., 1817 Mount Vernon Street.
1889 Deaver, H. C., 629 Oxford Street.
1885 Deaver, J. B., Demonstrator of Anatomy in the University of Pennsylvania, Surgeon to the Philadelphia Hospital, and to the German Hospital, 120 South Eighteenth Street.
1877 Deaver, R. W., resigned January 27, 1887.
1882 Dercum, F. X., Pathologist to the State Hospital for the Insane at Norristown, Pennsylvania; Chief of Dispensary for Nervous Diseases, University Hospital; Neurologist to the Philadelphia Hospital, 636 North Eighth Street.

ELECTED

1882 De Schweinitz, G. E., Ophthalmic Surgeon to the Children's Hospital and to the Philadelphia Hospital, 1401 Locust Street.
1885 Dock, George, resigned December 5, 1888.
1886 Donges, John W., Camden, New Jersey.
1888 Downs, Andrew J., 1218 North Eighteenth Street.
1889 Downs, Norton, Pennsylvania Hospital.
1877 Drysdale, T. M., 1531 Arch Street.
1866 Duer, Edward L., 1704 Arch Street.
1870 Duhring, L. A., resigned November 9, 1882.
1876 Dulles, C. W., 4101 Walnut Street.
1883 Dunn, T. D., West Chester, Pennsylvania. (N. R.)
1883 Dunmire, G. B., 1116 Arch Street.
1860 Dunton, W. R., 5059 Germantown Avenue, Germantown.
1863 Dyer, Ezra, Pittsburg, Pa. (N. R.)

1883 Edwards, J. F., forfeited membership by non-payment of dues.
1882 Edwards, W. A., San Diego, California. (N. R.)
1885 Egbert, J. C., General Wayne, Pennsylvania.
1891 Egbert, Seneca, 4808 Springfield Avenue.
1875 Engel, Hugo, forfeited membership by non-payment of dues, February 23, 1882.
1891 Eshner, A. A., 853 North Fifth Street.
1876 Eskridge, J. T., Colorado Springs, Colorado. (N. R.)

1881 Fenton, T. H., 1335 Arch Street.
1857 Fischer, Emil, 729 North Sixth Street. (O. M.)
1863 Fish, Augustine H., died August 3, 1872.
1876 Fisher, Henry M., Physician to the Episcopal Hospital, 919 Walnut Street.
1890 Flick, L. J., 736 Pine Street.
1857 Forbes, William S., forfeited membership by non-payment of dues.
1869 Ford, William H., 1622 Summer Street.
1878 Formad, H. F., Demonstrator of Pathology in the University of Pennsylvania, 3535 Locust Street.
1871 Fox, Charles, resigned December 10, 1884.
1888 Fox, J. M., Torresdale.
1886 Freeman, W. J., 2034 Chestnut Street.
1890 Freibis, George, 1720 Chestnut Street.

ELECTED

1880 French, M. S., 1437 Spruce Street.
1890 Fuchelis, P., 216 Franklin Street.
1885 Fussell, M. H., 168 Green Lane, Manayunk.

1868 Garretson, James E., resigned November 22, 1883.
1883 Garrett, E. F., 5103 Germantown Avenue, Germantown.
1871 Gerhard, George S., Ardmore, Pennsylvania. (N. R.)
1870 Getchell, Frank H., 1432 Spruce Street.
1890 Gibb, Joseph C., 817 Franklin Street.
1877 Gilliard, Louis, died November, 1878.
1890 Gleason, E. Baldwin, 1204 Walnut Street.
1882 Godey, H. E., N. E. corner Nineteenth and Spruce Streets.
1877 Goldsborough, C. B. (N. R.)
1866 Goodell, William, Clinical Professor of Diseases of Women in the University of Pennsylvania, 1418 Spruce Street.
1886 Goodell, W. C., 1418 Spruce Street.
1868 Goodman, H. Ernest, resigned February 23, 1882.
1868 Graham, James, resigned January 9, 1879.
1890 Grayson, C. P., 122 South Forty-second Street.
1887 Green, Walter D., 131 South Fifteenth Street.
1867 Grier, Matthew J., 1531 Spruce Street.
1883 Griffith, J. P. C., Assistant Physician to the University Hospital, 123 South Eighteenth Street.
1868 Gross, Ferdinand H., Surgeon to the German Hospital, 1416 Girard Avenue.
1885 Gross, Onan B., 700 Market Street, Camden, New Jersey.
1857 Gross, Samuel D., died May 6, 1884. (O. M.)
1858 Gross, Samuel W., died April 16, 1889.
1869 Grove, John H., resigned September, 1890.
1862 Gruel, Theodore H. E., 612 Spruce Street.
1874 Guitéras, John, Professor of Pathology in the University of Pennsylvania.
1890 Gummey, Frank B., Pennsylvania Hospital.

1884 Haehlen, W. F., 1421 North Broad Street.
1871 Hale, George J., forfeited membership by non-payment of dues, March 23, 1876.
1857 Hall, A. Douglass, resigned October 9, 1873. (O. M.)
1890 Hall, William S., Haverford College.
1886 Hamaker, W. D., Meadville, Pennsylvania. (N. R.)

ELECTED

1875 Hand, Frank C., died September 1, 1881.
1885 Hare, Hobart A., Professor of Therapeutics in the Jefferson Medical College, 222 South Fifteenth Street.
1866 Hare, Horace Binney, died March 21, 1879.
1868 Hargadine, R. W. (N. R.)
1859 Harlan, George C., resigned January 12, 1882.
1874 Harris, Charles M., died.
1858 Harris, Robert P., resigned January 9, 1873.
1890 Harrison, W. H., Pennsylvania Hospital for Insane.
1881 Hart, R. H., forfeited membership by non-payment of dues.
1857 Hartshorne, Edward, died June 29, 1885. (O. M.)
1859 Hartshorne, Henry, resigned October 12, 1864.
1890 Hartzell, M. B., 3725 Spring Garden Street.
1867 Hassler, Ferdinand A., resigned February 9, 1871.
1890 Hatch, John O., 3615 Locust Street.
1869 Hatfield, Nathan L., died.
1868 Hays, I. Minis, resigned January 13, 1881.
1879 Hazlehurst, S. F., Colorado Springs, Col. (N. R.)
1873 Hearn, Joseph, Surgeon to the Jefferson Medical College Hospital, and to the Philadelphia Hospital, 1120 Walnut Street.
1890 Heil, W. S. G., 4943 Lancaster Avenue.
1870 Henry, Frederick P., Physician to the Episcopal Hospital, and to the Philadelphia Hospital, 1635 Locust Street.
1866 Herbert, Theodore. (N. R.)
1879 Hess, Robert, resigned January 12, 1882.
1857 Hewson, Addinell, forfeited membership by non payment of dues, December 22, 1881. (O. M.)
1880 Hewson, Addinell, Jr., 350 South Sixteenth Street.
1874 Hickman, N. B., forfeited membership by non-payment of dues, December 8, 1881.
1890 Hinkle, F., Columbia, Pennsylvania.
1884 Hinsdale, Guy, resigned January, 1891.
1887 Hirst, Barton Cooke, Professor of Obstetrics in the University of Pennsylvania, 248 South Seventeenth Street.
1858 Hodge, H. Lenox, died June 10, 1881.
1883 Hoelly, G. H., forfeited membership by non-payment of dues.
1871 Hoffman, Washington Atlee, died September 20, 1874.
1886 Holland, J. W., resigned October 25, 1888.
1860 Hopkins, Henry St. George. (N. R.)
1876 Hopkins, W. B., resigned October 14, 1880.

ELECTED

1877 Horwitz, Theodore, died December 12, 1877.
1870 Houston, James P. S., Savannah, Georgia. (N. R.)
1891 Howard, C. E.
1866 Howe, Herbert M., resigned February 28, 1878.
1859 Hoyt, William D. (N. R.)
1880 Hughes, D. E., Philadelphia Hospital.
1882 Hughes, W. E., Physician to the Philadelphia Hospital, 3726 Baring Street.
1879 Huidekoper, R. S., resigned October 28, 1880.
1857 Humphries, George H., New York City. (O. M., N. R.)
1868 Hunter, Charles T., resigned October 14, 1880.
1870 Hutchins, E. R. (N. R.)
1858 Hutchinson, James H., died December 27, 1889.

1866 Ingham, James V., resigned December 11, 1879
1866 Ingram, T. D., forfeited membership by non-payment of dues. December 8, 1881.
1890 Izard, William, Camden, New Jersey.

1874 Jamison, Edward., St. Louis, Missouri. (N. R.)
1890 Jarrett, Henry, Camden, New Jersey.
1866 Jenks, William, died October 31, 1881.
1875 Johnson, Russell H., resigned December 8, 1881.
1891 Jones, Charles J., 1720 North Nineteenth Street.
1883 Judd, L. D., 3603 Powelton Avenue.
1885 Jurist, L. J., 839 North Broad Street.

1859 Kane, John K., Wilmington, Delaware. (N. R.)
1857 Kealer, William, resigned January 28, 1875. (O. M.)
1874 Keating, John M., 1504 Walnut Street.
1857 Keating, William V., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)
1866 Keen, William W., resigned December 9, 1886.
1875 Kelly, Joseph V., 4257 Main Street, Manayunk.
1890 Ketcham, S. Rush. (N. R.)
1884 Kilduff, Robert, resigned December 9, 1886.
1889 Kimmell, Louis, J. C., died 1890.
1858 King, William, died.
1888 Kirby, Ellwood R.
1879 Kirkbride, J. J., resigned December 13, 1888.

ELECTED

1890 Kneass, Samuel S., University Hospital.
1869 Knight, Samuel R., resigned October 28, 1880.

1867 Lambdin, A. C., forfeited membership by non-payment of dues, December 10, 1874.
1871 Landis, Henry G., died.
1890 La Place, Ernest, Visiting Surgeon to Philadelphia Hospital, 1617 Arch Street.
1872 La Roche, C. Percy, Nice, France. (N. R.)
1857 La Roche, René, died December 9, 1872. (O. M.)
1879 Leach, A. L., forfeited membership by non-payment of dues, December 22, 1881.
1869 Leaman, Henry, 828 North Broad Street.
1860 Lee, Charles C., New York City. (N. R.)
1882 Lee, J. G., resigned September, 1883.
1861 Leedom, John M., died.
1887 Leopoldt, Isaac, 1520 Franklin Street.
1857 Levick, James J., resigned October 13, 1861. (O. M.)
1868 Lewis, Francis W., 2016 Spruce Street.
1869 Lewis, Fred. W., died 1873.
1885 Lewis, George T., resigned December 13, 1888.
1875 Lewis, Morris J., Physician to the Episcopal Hospital, 1216 Walnut Street.
1857 Lewis, Samuel, resigned December 13, 1888. (O. M.)
1891 Lincoln, William R., 1723 North Eighteenth Street.
1877 Little, William S., died February 17, 1887.
1859 Livezy, Edward, forfeited membership by non-payment of dues, September 14, 1864.
1886 Lloyd, James Hendrie, Neurologist to the Philadelphia Hospital, 200 South Fortieth Street.
1876 Loder, P. E., forfeited membership by non-payment of dues, February 9, 1882.
1886 Longaker, D., 652 North Eighth Street.
1873 Longenecker, Jerome, forfeited membership by non-payment of dues, 1875.
1875 Longstreth, Morris, Physician to the Pennsylvania Hospital, 1416 Spruce Street.
1870 Loughlin, J. Eneu, forfeited membership by non-payment of dues, May 9, 1878.

ELECTED

1882 MacConnell, W. G., forfeited membership by non-payment of dues.

1873 MacCoy, A. W., 1415 Walnut Street.

1890 Mallet-Prevost, W. (N. R.)

1874 Mann, Charles H. (N. R.)

1867 Markoe, James, died March, 1884.

1887 Martin, Edward, 415 South Fifteenth Street.

1868 Martin, George. (N. R.)

1874 Matteson, Charles C., died.

1865 Maury, Frank F., died 1879.

1870 Maxwell, J. G., forfeited membership by non-payment of dues, December 10, 1874.

1866 McArthur, J. A., forfeited membership by non-payment of dues, December 8, 1881.

1861 McCall, C. A., resigned.

1880 McClellan, G., 1352 Spruce Street.

1868 McClure, W. W., resigned September 10, 1878.

1884 McIlvaine, E. S., resigned December 11, 1884.

1890 McKelway, George I., Obstetrician to Philadelphia Hospital, 255 South Seventeenth Street.

1864 Mears, J. Ewing, Surgeon to St. Mary's Hospital, 1429 Walnut Street.

1873 Meigs, Arthur V., Physician to the Pennsylvania Hospital, 1322 Walnut Street.

1857 Meigs, John Forsythe, died December 16, 1882. (O. M.)

1874 Miller, C. K. I., died 1878.

1882 Miller, D. J. M., 238 South Twenty-first Street.

1878 Mills, C. K., Professor of Diseases of the Mind and Nervous System in the Philadelphia Polyclinic and School for Graduates, Neurologist to the Philadelphia Hospital, 1909 Chestnut Street.

1885 Mitchell, James, forfeited membership by non-payment of dues.

1884 Mitchell, J. K., Physician to St. Mary's Hospital, Assistant Physician to the University Hospital, 211 South Seventeenth Street.

1857 Mitchell, S. Weir, resigned. (O. M.)

1876 Montgomery, E. C., resigned January 11, 1883.

1857 Morehouse, George R., 2033 Walnut Street. (O. M.)

1885 Morrison, William H., Holmesburg, Pennsylvania. (N. R.)

1887 Morton, S. W., 318 South Fifteenth Street.

ELECTED

1857 Morton, T. G., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)
1885 Morton, T. S. K., resigned January 1, 1891.
1859 Moss, William, resigned December 10, 1885.
1871 Muhlenberg, Frank, Reading, Pennsylvania. (M. R.)
1869 Muller, August F., 4753 Green Street, Germantown.
1880 Musser, J. H., Physician to the Philadelphia Hospital, Pathologist to the Presbyterian Hospital, Chief of Medical Dispensary University Hospital, 229 South Fortieth Street.
1873 Musser, M. B., died March, 1888.
1869 Mustin, J. Burton, died 1871.
1890 Mutchlock, G. A., 848 Wharton Street.

1869 Nancrede, C. B. (N. R.)
1879 Neff, J. S., 1425 Spruce Street.
1885 Neilson, T. R., Surgeon to the Episcopal Hospital, 223 South Seventeenth Street.
1880 Newcomet, H. W., died June 1, 1885.
1872 Newsham, Stanley P., resigned December 23, 1881.
1868 Norris, Herbert, resigned January 8, 1880.
1868 Norris, Isaac, resigned November 12, 1874.
1869 Norris, J. C., resigned February 10, 1876.
1861 Norris, William F., Clinical Professor of Ophthalmology in the University of Pennsylvania, 1530 Locust Street.

1875 O'Hara, Michael, Physician to St. Mary's Hospital, 31 South Sixteenth Street.
1891 O'Hara, Michael, Jr., 31 South Sixteenth Street.
1886 Oliver, C. A., resigned November 20, 1887.
1874 Osgood, Hamilton. (N. R.)
1885 Osler, William, Baltimore, Maryland. (N. R.)

1885 Packard, F. A., 131 South Fifteenth Street.
1857 Packard, John H., Surgeon to the Pennsylvania Hospital, 1926 Spruce Street. (O. M.)
1868 Pancoast, William H., Professor of Surgical Anatomy and Clinical Surgery in the Medico-Chirurgical College, 1100 Walnut Street.
1873 Parish, William H., resigned December 8, 1887.
1875 Parks, Edward L. (N. R.)
1867 Parry, John S., died 1876.

ELECTED

1870 Paul, Comegys, Belvidere, New Jersey. (N. R.)
1875 Paul, James, resigned January 22, 1880.
1890 Pennock, W. J., 1407 North Seventeenth Street.
1891 Penrose, Charles B., 1331 Spruce Street.
1857 Penrose, R. A. F., forfeited membership by non-payment of dues, September 14, 1864. (O. M.)
1865 Pepper, George, died September 14, 1872.
1865 Pepper, William, Professor of Theory and Practice of Medicine in the University of Pennsylvania, 1811 Spruce Street.
1879 Perkins, F. M., resigned December 28, 1888.
1889 Pfouts, Gilbert B., Salt Lake City, Utah. (N. R.)
1885 Piersol, G. A., Chester Avenue and Forty-eighth Street.
1870 Porter, William G., resigned December 28, 1888.
1890 Potts, C. S., 1712 Wallace Street.
1886 Powell, William M., 2205 Pine Street.
1890 Prendergast, W. T., 2332 Montgomery Avenue.
1889 Price, Joseph, 500 North Twentieth Street.

1885 Rahter, C. A., Harrisburg, Pennsylvania.
1885 Randall, B. A., Ophthalmic Surgeon to the Children's Hospital, 1806 Chestnut Street.
1878 Raub, J. A., forfeited membership by non-payment of dues.
1885 Reed, Boardman, Atlantic City, New Jersey.
1884 Reed, C. H., resigned December 22, 1887.
1874 Reed, H. B., resigned December 9, 1880.
1859 Reed, Thomas B., died 1891.
1871 Rex, George A., 2118 Pine Street.
1869 Rex, O. P., forfeited membership by non-payment of dues, December 10, 1874.
1877 Reynolds, A. S., forfeited membership by non-payment of dues, February 9, 1882.
1864 Rhoads, Edward, died January 16, 1871.
1878 Rich, T. C., resigned January 13, 1881.
1869 Richardson, Elliott, resigned December 8, 1881.
1863 Richardson, Joseph G., died November 13, 1886.
1857 Richardson, T. G., New Orleans, Louisiana. (O. M., N. R.)
1884 Ridge, J. M., Camden, N. J.
1886 Riera, J. H., 1630 Vine Street.
1876 Risley, S. D., Lecturer on Ophthalmoscopy in the University of Pennsylvania, 1722 Walnut Street.

ELECTED

1868 Ritz, Charles N. (N. R.)

1886 Riveley, M. P., 121 South Eighteenth Street.

1880 Roberts, A. S., Instructor in Orthopædics in the University of Pennsylvania, 1901 Walnut Street.

1876 Roberts, J. B., Professor of General and Operative Surgery in the Philadelphia Polyclinic and School for Graduates, Surgeon to St. Mary's Hospital, 1627 Walnut Street.

1889 Robinson, William Duffield, 2012 Mt. Vernon Street.

1876 Roland, O. (N. R.)

1874 Ronaldson, W. D. (N. R.)

1877 Rudolph, B. J., forfeited membership by non-payment of dues, February 22, 1882.

1867 Santee, Eugene J., 605 North Eleventh Street.

1890 Sartain, Paul J., 212 West Logan Square.

1863 Savery, William, resigned February 11, 1869.

1869 Schell, H. S., resigned December 8, 1881.

1874 Seiler, Carl, 1346 Spruce Street.

1870 Seyfert, T. H., resigned December 27, 1887.

1870 Shaffner, Charles, 251 South Thirteenth Street.

1876 Shakespeare, E. O., Pathologist to the Philadelphia Hospital, Ophthalmic Surgeon to the Philadelphia Hospital, 1336 Spruce Street.

1869 Shapleigh, Elisha B., 658 North Eighth Street.

1868 Sherwood, T. H., forfeited membership by non-payment of dues, December 10, 1874.

1885 Shober, J. B., 1311 Spruce Street.

1889 Shoemaker, Harvey, 109 South Twentieth Street.

1878 Shoemaker, J. V., 1519 Walnut Street.

1871 Simes, J. H. C., Professor of Genito-urinary Diseases in the Philadelphia Polyclinic and School for Graduates, Surgeon to the Episcopal Hospital, and to St. Christopher's Hospital, 2033 Chestnut Street.

1883 Simon, J. W. (N. R.)

1868 Sinkler, Wharton, Physician to the Hospital for Nervous Diseases, 1606 Walnut Street.

1881 Skillern, P. G., 429 South Broad Street.

1881 Skillern, S. R., 3509 Baring Street.

1890 Sleifer, North Wales, Pennsylvania.

1890 Smith, A. Donaldson, 222 South Twenty-first Street.

ELECTED

1858 Smith, A. H., died December, 1885.
1887 Smith, Allen J., 332 South Seventeenth Street.
1863 Smith, E. A. (N. R.)
1859 Smith, F. G., resigned November 13, 1873.
1876 Smith, R. M., resigned December 9, 1880.
1875 Smith, Stanley. (N. R,)
1876 Smythe, F. G., died 1879.
1871 Sparks, G. W., forfeited membership by non-payment of dues, December 10, 1874.
1866 Spooner, E. A., forfeited membership by non-payment of dues, December 10, 1874.
1871 Starr, Louis, resigned October 14, 1880.
1889 Stevens, Arthur A., 4656 Green Street, Germantown.
1886 Stewart, David D., 2628 North Fifth Street.
1883 Stiles, G. A., Conshohocken, Pennsylvania.
1857 Stillé, Alfred, Professor Emeritus of the Theory and Practice of Medicine in the University of Pennsylvania, 3900 Spruce Street. (O. M.)
1870 Strawbridge, George, resigned June 10, 1875.
1884 Stritmatter, I. P., 1232 North Fifth Street.
1869 Stryker, Samuel S., 3821 Walnut Street.
1889 Sudduth, W. H., 1215 Filbert Street.

1891 Taylor, H. W.
1879 Taylor, J. M., 1504 Pine Street.
1886 Taylor, L. H., Wilkesbarre, Pennsylvania. (N. R.)
1866 Thomas, C. H., resigned February 11, 1886.
1868 Thomson, William, Surgeon to Wills Ophthalmic Hospital, 1426 Walnut Street.
1870 Tinkham, J. H., United States Navy. (N. R.)
1866 Townsend, George D. (N. R.)
1868 Townsend, R. M., died.
1886 Tull, M. G., resigned December 6, 1886.
1864 Turner, A. P. (N. R.)
1890 Turner, John B., 724 South Sixteenth Street.
1863 Tutt, Charles Pendleton, died May 11, 1866.
1863 Tyson, James, Professor of Clinical Medicine in the University of Pennsylvania, 1506 Spruce Street.
1890 Tyson, J. Mellor, 1506 Spruce Street.

ELECTED

1885 Ulrich, W. B., Chester, Pennsylvania, forfeited membership by non-payment of dues.

1871 Valdivieso, R. (N. R.)

1870 Van Harlingen, Arthur, resigned December 8, 1881.

1887 Vansant, Eugene L., Physician to the Philadelphia Hospital, 113 South Sixteenth Street.

1879 Van Valzah, W. W., resigned.

1887 Walker, J. B., Obstetrician to the Philadelphia Hospital, 1617 Green Street.

1891 Wallace, James, 123 North Sixteenth Street.

1890 Walter, Charles, died 1891.

1890 Warden, Charles B., 1401 North Sixteenth Street.

1874 Warder, W. H., Obstetrician to the Philadelphia Hospital, 1212 North Broad Street.

1867 Watson, E. W., forfeited membership by non-payment of dues.

1866 Weightman, J. F., resigned April 27, 1871.

1876 Weir, A. H., resigned April 14, 1881.

1889 Wells, George M., General Wayne, Pennsylvania. (N. R.)

1875 West, Franklin, died.

1887 Westcott, T. S., 28 South Seventeenth Street.

1882 Wetherill, H. M., resigned October, 1885.

1885 Wetherill, R. B., Lafayette, Indiana. (N. R.)

1885 Wharton, H. R., Surgeon to the Cnildren's Hospital, and to the Presbyterian Hospital, Demonstrator of Surgery in the University of Pennsylvania, 112 South Eighteenth Street.

1875 Whelen, Alfred, resigned December 23, 1880.

1873 White, J. William, Professor of Clinical Surgery in the University of Pennsylvania, 1806 Locust Street.

1868 Willard, De Forest, Lecturer on Orthopædic Surgery in the University of Pennsylvania, Surgeon to the Presbyterian Hospital, 1818 Chestnut Street.

1866 Williams, Horace, resigned December 11, 1879.

1890 Williams, M. H., 868 North Eighth Street.

1875 Williamson, Jesse, resigned October 13, 1881.

1889 Wilmuth, A. W., Elwyn, Pennsylvania. (N. R.)

1882 Wilson, C. M., 121 South Seventeenth Street.

1867 Wilson, Elwood, resigned.

1880 Wilson, H. A., resigned December 30, 1882.

ELECTED

1869 Wilson, James C., Physician to the Jefferson Medical College Hospital, 1437 Walnut Street.

1867 Wilson, James F., 1010 Race Street.

1869 Wilson, J. H. (N. R.)

1891 Wilson, Samuel M., 1010 Race Street.

1891 Wilson, W. R., 1633 Locust Street.

1873 Winslow, W. H., resigned December 28, 1876.

1864 Wistar, Thomas, resigned February 13, 1867.

1875 Wolford, W. S., forfeited membership by non-payment of dues.

1890 Wolff, L., 333 South Twelfth Street.

1890 Wood, A. C., 1205 Spruce Street.

1863 Wood, H. C., Jr., forfeited membership by non-payment of dues, March 23, 1876.

1883 Woodbury, F., 218 South Sixteenth Street.

1865 Woods, D. Flavel, Physician to the Presbyterian Hospital, 1501 Spruce Street.

1853 Woodward, J. J., U. S. A., died 1884. (O. M.)

1872 Worthington, D. J., forfeited membership by non-payment of dues, February 9, 1882.

1886 Young, J. K., Surgical Registrar to the University Hospital, 220 South Sixteenth Street.

1876 Ziegler, G. W. (N. R.)

CORRESPONDING MEMBERS.

ELECTED

1866 Bumstead, Freeman, J. Died 1879.

1859 Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York.

1859 Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York.

1885 Dent, Clinton T., Assistant Surgeon and Lecturer on Practical Surgery at St. George's Hospital, Surgeon to Belgrave Hospital for Children, London, England.

1860 Ellis Calvin. Died 1883.

1888 Fedeli, Gregorio, Rome, Italy.

1858 Flint, Austin, Sr. Died 1886.

1858 Hammond, William A., member of the National Academy of Sciences, New York.

1860 Isaacs, Charles E., Brooklyn. Died June 16, 1860.

1878 Jackson, J. B. S., Boston. Died 1879.

1886 Pye-Smith, P. H., Physician and Lecturer on Medicine at Guy's Hospital, London, England.

1860 Reeves, James E., Wheeling, West Virginia.

1861 Rokitansky, Carl. Died 1878.

1860 Watson, John, New York. Died June 3, 1869.

1859 Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

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REPORT.

I. THE OSSEOUS SYSTEM.

1. *Specimens showing disease of the knee-joint.*

Exhibited by Dr. H. R. Wharton.

The first specimen, showing disease of the knee-joint, was removed from a patient in the Children's Hospital. In this case amputation of the thigh was resorted to, as the destruction of the tissues about the knee-joint was so extensive that excision was not considered advisable.

The history of the case is as follows:

John McQ., æt. 2½ years, was admitted to the Children's Hospital, June 15, 1885. At the time of his admission he was suffering from strumous disease of the right knee and elbow-joints, he had also had mastoid disease. Several months after his admission he was attacked with croup, and as his dyspnœa became urgent I performed tracheotomy, and he made a good recovery. A few months afterward a large sequestrum was removed from his right mastoid region.

The patient also had his right elbow excised, and made a good recovery.

Later, the disease in the right knee-joint progressed so actively, and the soft parts about the knee became involved to such an extent, that it was considered advisable to remove the diseased structures by amputation of the thigh, instead of attempting an excision of the joint. The specimen removed by this operation I show this evening.

After he had recovered from his amputation, a swelling which he had on the right side of the scalp, over the parietal bone, became ulcerated, and exposed the surface of the skull. The patient at this time had convulsions, and was paralyzed on the left side. He was then trephined over the seat of the exposed bone, and an intracranial abscess was evacuated, after which his convulsions and paralysis disappeared. The

patient did well for ten days, then had several convulsions, became comatose, and died.

I present also the knee-joint of Mary D., æt. 12 years, who was a patient in the Children's Hospital, which presents extensive disease of the knee-joint. In this case excision of the joint was attempted, but on exposure of the joint surfaces it was found that the disease of the bone was so extensive that it was considered advisable to remove the diseased structures by amputation of the thigh. *December* 22, 1887.

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2. *Dactylitis syphilitica.*

Exhibited by Dr. J. K. Young.

F. B., æt. 32 years, German, grocer's clerk by occupation. One and a half years ago his phalangeal joint became swollen from knocking his hand on the table excessively in card-playing. Small abscesses formed, which he opened, and were followed by sinuses. He denied syphilitic history, but during his stay in the hospital he developed a pneumonia which was considered specific.

Cases of dactylitis following acquired syphilitis, in adults are much rarer than those which occur early as a sequence of hereditary infection. The latter form of the disease was brought before the profession by Dr. R. W. Taylor, of New York, in the *American Journal of Syphilography and Dermatology*, in January, 1871. He believes that "Dactylitis is usually observed in very young children, but it may occur as late as in the twentieth year."

Dr. F. Eschele (*Archiv für klin. Chirurgie*, vol. xxxvi.) has recently called attention to this disease. He believes that "gummatous growths in the medulla and periosteum, as well as gummata of the neighboring parts, are included under this title; that in adults the periosteal form is most common, and believes that traumatism is of minor importance."

The specimen presented shows few lesions of the joint, considering the long time the disease has existed. The articulating joint surfaces are denuded of their cartilage, but otherwise remain unchanged. The periosteum about the joint everywhere is much thickened and indurated. This is as we should expect to find it. *February* 29, 1888.

3. *Dactylitis.*

Exhibited by Dr. F. A. Packard.

The specimen was obtained from a colored child, æt. 3 years, who was brought to the hospital three or four nights ago on account of free hemorrhage from a bunch of granulations over the middle phalanx of the index finger. The phalanx itself was swollen, and the bones evidently thickened. The granulations had ulcerated through the skin, and a probe passed down touched bare bone. The finger was amputated March 7, 1888, by Dr. Ashhurst. On dissection the skin is found to have been perforated by a granulation mass springing from the enlarged middle phalangeal bone. The mother gave a history of two miscarriages, both after the birth of this child. No other cause to suspect syphilitis. Teeth normal. The child had sweating about the head at night, but no other evidences of struma. There was also found a swelling of the left breast, said to be of two weeks' duration—but doubtully so—that was cystic, and was probably a galactocele, although no milk exuded on pressure. *March* 8, 1888.

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4. *Caries of head and neck of humerus.*

Exhibited by Dr. H. R. Wharton.

The patient from whom this specimen was removed was a man æt. 34 years, who had six weeks previously suffered from pain in the upper part of the left arm, which in a few weeks was followed by the formation of a large abscess. This abscess continued to discharge, and the arm became useless. Examination with a probe showed carious bone in the position of the head of the humerus.

The head and neck of the humerus were excised, and were, as the specimen shows, in a softened and carious condition; the head of the bone was also surrounded by a purulent collection.

September 27, 1888.

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5. *Caries of atlas and axis.*

Exhibited by Dr. J. Daland.

Patrick L., æt. 70 years, about one year ago, while lifting a heavy iron rail, felt something give way in the neck and the head fell backward and then forward. He resumed work, and after some weeks

he began to complain of stiffness and pain in the neck. Pain was also complained of in the head, and soon repeated crops of herpes zoster showed themselves. He grew worse, and gradually the arms and legs became paralyzed. There was no interference with the functions of the bladder or rectum. It was now impossible for him to rotate his head to the right or left, but he still could move it backward and forward to a slight degree. He remained in this condition for some months, when a retropharyngeal abscess formed, which was lanced, liberating considerable pus. In a short time the paralysis disappeared, and he recovered full control of the muscles of the arms and legs, but he was never able to hold his head erect. He died from exhaustion, and a post-mortem examination showed considerable caries of the atlas and axis.

It seems clear that in the beginning he ruptured one of the ligaments, and that the inflammation then extended to the bone.

The occurrence of herpes zoster reminds us of the well-known relation to organic disease. The relief afforded by the escape of pus from lancing the post-pharyngeal abscess would seem to support the idea that the paralysis was simply due to pressure. *November* 22, 1888.

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6. *Caries of fourth and fifth lumbar vertebræ.*

Exhibited by Dr. J. Daland.

T. M., æt. 32 years, a hat-maker by occupation. He is a widower, and has no children. His family history is very good, and he has always enjoyed good health until six years ago, when he passed through a mild attack of uncomplicated typhoid fever. There is no evidence of syphilis nor scrofula.

In January, 1888, his health began to fail, and he complained of dyspepsia, diarrhœa alternating with constipation, loss of flesh, strength, and color. These symptoms became more pronounced after the death of his wife, in April, 1888.

The first symptoms of vertebral disease showed itself in May, 1888, when he began to suffer from paroxysmal attacks of moderately severe, knife-like pain in the left sciatic, which gradually increased in intensity until it became continuous. At first the pain was confined to the left thigh, but later it extended to the left knee and foot, and soon involved the right leg.

About this time it was observed that there was considerable loss of power in the legs, and that the left leg was more wasted than the right.

Transient œdema of the feet occurred in September, and the left was much more swollen than the right. The heart and kidneys were examined and found normal.

In October, or five months after the first development of sciatic pain, his bladder and rectum became paralyzed. There was no chill, fever, sweat, nor herpes. He died in coma, November 5, 1888.

At the autopsy, which was performed the next day, a slight swelling was observed in the left iliac fossa, and, upon making an incision through the psoas muscle, about five ounces of pus and broken-down tissue escaped. Careful examination showed that the left side of the bodies of the fourth and fifth lumbar vertebræ were destroyed to such an extent that the finger could readily pass into the spinal canal. The other organs showed nothing of interest. *November* 22, 1888.

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7. *A unique case of multiple exostoses.*

Exhibited by Dr. J. Leidy, Jr.

The following interesting case of multiple exostoses presented herself at the surgical clinic of the University Hospital, with the following history:

Margaret P., æt. 25 years, housemaid by employment, born in Ireland, with a good family and personal history, comes complaining of severe pain in the left thigh and both knees, due to the presence of several large hard tumors in the corresponding positions. She not only suffers pain, but at times is unable to maintain her equilibrium, necessitating the separation of her feet for some distance so as to give her a broad base upon which to stand.

The largest growth, about the size of a cocoanut, prevents her maintaining the recumbent or sitting posture, without experiencing severe pain. The only relief is to be obtained while in the erect position. Her powers of locomotion are considerably interfered with by several growths upon the inner side of the knees. The former outgrowth she first discovered ten years ago, and she believes it to be increasing in size. The existence of the above multiple tumors, of bony consistence, upon the same individual, led to a careful examination of the whole body, which showed the presence of hard bony outgrowths varying from the size of a cocoanut to a grain of corn, to the number of 126, arranged symmetrically upon the following bones:

The largest outgrowths were found upon the following: Right and

left femur, tibia, and fibula, pelvis, left scapula, seventh, eighth, ninth, and tenth left ribs anteriorly, sixth, seventh, and eighth ribs posteriorly. Growths of variable sizes were found upon the metatarsal and carpal bones, phalanges, ulna, humerus, clavicle, ribs, ilium, and ischium. Upon the shafts of several of the long bones were elevations which conveyed the sensation as though shot had been imbedded beneath the periosteum. The bones of the skull were not affected.

The above outgrowths, variable in size and shape, were all of stony hardness, both pediculated and sessile, and smooth and irregular upon the surface.

Pain, which was the principal symptom in this case, was purely mechanical. Deep pressure failed to elicit any marked tenderness; friction of the superficial tissues, on the other hand, gives rise to severe pain, which radiates from the growth principally along the course of the nerves in close proximity. The pain in the knees was clearly demonstrated as due to interference.

The history of these growths is not without interest. As is usually the case, they occurred insidiously without the patient's knowledge. With the exception of the one upon her thigh, she was unconscious of their existence until her attention was drawn to them. It appears almost inconceivable how such a growth, the size of that upon the scapula or the phalanges, could have existed for any length of time without the patient's knowledge.

Several of the largest outgrowths are of sufficient interest to deserve a detailed description:

The left scapula was so distorted by an irregular growth, the size of two fists, which arose from the body of the bone, that its normal contour was completely destroyed. The prominence upon the left femur consisted of a large bony outgrowth, of stony hardness, hemispherical in shape, the semicircumference of which measured ten inches, perfectly smooth upon its surface, and attached by a broad base to the posterior aspect of the shaft of the femur, just below the trochanter. The projections from the inner condyles of both femurs and upon the tibiæ and fibulæ, mostly coracoïd in shape, and irregular upon their surface, extended in such a direction as to interfere markedly with the movement of the lower limbs during the act of walking. Bursæ were to be found over several of the above prominences.

The character and position of the above osseous masses, connected as they are with the epiphyses, determine them as belonging to the cancellous variety of the so-called epiphyseal exostoses by development, though we would mention in this connection their extension from the

epiphyses and along the shafts of several of the long bones. In this respect they rather conform to that variety to which Poncet has applied the term juxta-epiphyseal exostoses, and such classification appears to be anatomically the more accurate.

The etiology of multiple exostoses is little understood, the absence of inheritance, tuberculosis, syphilis, gout, rheumatism, cancer, in fact, any variety of tumor as an etiological factor makes it impossible to give an adequate explanation as to the cause in this case.

Showing as this case does such an unusual disposition to the formation of osseous tissue disposes us to classify it among the diatheses; we could obtain no better example of the so-called osteogenic diathesis. On the other hand, there are those who are inclined to view such cases as instances of monstrosities by excess.

Though cases of multiple exostoses are not rare, the above case is unique as regards the unusual number of outgrowths and the number of different bones affected. The nearest approach we can find was in a lad aged ten years, upon whom Virchow found sixty-five exostoses, following an attack of acute articular rheumatism.

In conclusion, the writer wishes to express his thanks and indebtedness to Dr. Agnew for the privilege of placing the above case on record.

November 22, 1888.

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8. *Small tumor removed from the internal condyle of the femur.*

Exhibited by Dr. H. R. Wharton.

R. M., æt. 54 years, during the War of the Rebellion received a fall upon the right knee, and noticed shortly afterward a swelling over the pre-patellar bursa. This swelling remained stationary for some years and caused him no pain. Since 1884 the swelling has increased much in size and has become painful. In July of 1888, he presented himself at the University Hospital, and it was then found that a tumor, the size of a small orange, occupied the position of the pre-patellar bursa. The tumor was cut down upon and seen to be a solid growth. In order to remove it completely, it was necessary to open the knee-joint freely. The joint was drained and the wound closed. The patient made a good recovery. During the early part of this year the patient again presented himself at the hospital, complaining of pain at the inner side of the knee-joint, and a careful examination revealed a small growth occupying the position of the inner condyle of the femur, and firmly attached

to it. This was incised and found to be a mass of firm tissue, apparently having its origin in the cancellous structure of the inner condyle of the femur. It was removed by means of a gouge, and upon examination presented the gross appearances of a sarcoma. The case is of interest in view of the removal previously of the solid tumor from the position of the pre-patellar bursa, which was unfortunately mislaid so that a microscopical examination could not be obtained. Both growths are probably sarcomatous in their nature, although the slow development of the first growth is remarkable if such be the case, and it also occupied a very unusual position for a growth of this nature.

February 28, 1889.

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9. *Pre-patellar bursal tumor.*

Exhibited by Dr. H. R. Wharton.

T. S. R., æt. 26 years, first noticed some swelling below the knee (left) three years ago, after receiving a blow on the knee in stepping from a wagon. The swelling enlarged, and formed a well-marked tumor, about the size of a man's fist, at the position of the pre-patellar bursa. The tumor was removed by a careful dissection and it was found that it did not involve the joint. Before its removal a mass resembling a loose piece of cartilage could be felt at the base of the tumor. The growth represents a form of tumor often seen in this locality, known as enlarged pre-patellar bursa, or housemaid's knee.

February 28, 1889.

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10. *A study of acute infectious processes in bone.*

Read by Dr. Roswell Park.

With the introduction of modern methods of biological research, the study of surgical pathology has made a vast stride from the domain of empiricism toward that of true science. It it not too strong a statement if one claims that, in the light of results obtained from bacteriological researches, our whole literature of general and surgical pathology must be rewritten, or so much of it as was published previous to 1885. I do not mean hereby to assume that our present position is in many respects an absolutely stable one, but that our knowledge gained previous to 1885

has been so enriched and extended that it must enjoy a new presentation in order fairly to represent the subject.

Let me not appear, while making such assertions, in an attitude too easily assailed. I am not in sympathy with the class of enthusiasts who are represented (and perhaps misrepresented) as considering bacteriology and pathology as synonymous, or who hold to *bacterio-centric* views, if I may coin the term. Nevertheless, I feel that we stand to-day where we can state without succesful contradiction that the introduction of bacteriological studies and methods has revolutionized the principles, as well as the practice, of surgery, and that to their general acceptance is due its *renaissance*.

And now, with this general introduction, permit me to ask attention to a class of surgical diseases whose acquaintance was long since made by the clinician, but whose intimate nature, whose etiology and pathology, it has remained for the bacteriologist to clear up. I allude particularly to acute suppurative periostitis and acute infectious osteomyelitis—the names by which they are best known in this country. Terribly severe in their typical forms, often fatal, bringing dread and despair to patient and surgeon alike, causing the one to endure the torments of the damned, and the other often to curse the utter helplessness of his measures, they have been at once the enigma and the stigma of surgery.

But this is no longer the case. Their etiology is now well known; the numerous changes which take place both inside and outside of the bone are pretty well understood, and only the connecting links between easily-recognized trains of events are now concealed from our appreciation. And all this has been made possible by modern bacteriological and microscopical technique.

Acute osteomyelitis and acute suppurative periostitis are, like tuberculosis, erysipelas, anthrax, and numerous other diseases, infectious maladies which develop under the influence and after the introduction into the system of one or more definite microörganisms. For manifestation of their pathogenic activities they undoubtedly need a predisposing condition of the system, but they are themselves the determining causes. For the recognition of this last fact we are indebted to the bacteriologists. For a demonstration, or further evidence, that they enter through some open pathway, through some lesion revealed or concealed, we must needs study our cases more carefully. We must realize that these conditions may have their origin, for instance, from an ulcerated tooth, or from some other lesion equally easily overlooked.

It is my desire to direct attention to the pathological, rather than the

clinical or therapeutical aspect of my subject; and, furthermore, not so much to matters of gross pathology as to the most recent investigations into the truly parasitic nature of these diseases. To this end, let me at once take the position that without the presence of pyogenic bacteria we have no pus; a position which to-day needs no defence.

With this statement for a foundation, it shall be our task to try to make clear the biological position of the parasites to whose pernicious activity these disturbances are due, and to bring out such facts regarding their ports of entry, methods of development, and habits, as time may allow.

Questions to which we particularly need unevasive answers, are:

I. Is there a specific coccus of infectious osteomyelitis?

II. Can more than one microbe produce it?

III. How does any bacterium which can cause it effect an entrance into the system?

IV. What influences do previously existing diseases exert?

V. Is there possibility of a mixed infection in these cases, and in what does it consist?

Before attempting to answer these questions, let us secure a little help from a brief historical sketch of the matter.

History.—As Ivanoff has put it, "Between 'growing pains,' so called, which disappear after a few hours of repose, and the intensely agonizing acute osteomyelitis, which has been so justly styled *typhus des membres*, there are very wide differences." Under this name, acute osteomyelitis, more than one form of lesion has been described, while to the milder form different writers have given a variety of names. Schutzenberger, in 1853, was perhaps the first to call attention to this disease under the name of *rheumatic periostitis*, but during the same year Chassaignac carefully studied the disease and gave it the name by which it is now known. He presented to the Academy of Sciences a classic memoir upon this subject, and, in 1854, another to the Society of Surgery upon "Acute Subperiosteal Abscesses." While he recognized a wide distinction between them, he saw also that the two diseases presented certain points in common. In 1858 Becker described the same malady under the name of *phlegmonous periostitis*. Almost at the same time Klose published thirteen cases of this character under the name of *meningo-osteo-phlebitis*. In these cases he observed epiphyseal separation. About the same time, too, Gosselin published a memoir and described the disease under the name of *epiphyseal ostitis of adolescence*. In 1862 Gamet published a thesis which he entitled "Juxta-epiphyseal Osteo-periostitis." Others also gave it yet other names, among which was that of Giraldes,

who usually spoke of it as phlegmonous periostitis. In 1878 Lannelongue published his classical memoir, describing it under the name of acute osteomyelitis of adolescence. It is rather with the acute and suppurative form that we have to deal, consequently I will not follow the history of the recognition and identification of the subacute or more chronic forms.

The gravity of the disease has long attracted the attention of surgeons. Searching for the causes thereof, Recklinghausen, Klebs, and others, examining the different organs of patients dying from the disease, found, in 1874, a micrococcus which they met with in the muscles, heart, pericardium, kidneys, and elsewhere, and which they considered the pathogenic agent, and upon whose recognition they founded various theories—some considering it the specific cause of the disease, others denying it.

Kocher attempted in a systematic, experimental manner to establish the parasitic origin of the disease. He injected various foul and putrid materials, and sometimes succeeded in setting up the trouble, and sometimes he failed. His materials, however, were not derived from pure cultures. Some of them, therefore, must have contained the specific microbes by accident. His conclusions were suggestive rather than conclusive.

In 1880 Pasteur, examining pus from a case of this kind, found also micrococci, which he cultivated, but with which he did not experiment upon animals. In 1881 Schüller, examining a limb amputated on this account, found the same organism in the bone, the periosteum, and the articular cartilages; but Becker was perhaps the first, in 1883, to report complete investigations with these organisms. He isolated and cultivated them upon various media, and then experimented with them upon animals. Injections of these cultures into the abdominal cavity produced peritonitis, but no trace of the lesion. He then made various contusions and fractures subcutaneously, and afterward injected these cultures into the veins; by this means he evoked the ordinary anatomical signs of osteomyelitis. He found them also in the lungs and kidneys, and concluded that he had to deal with a specific cause for the disease. Rosenbach repeated the experiments of Becker and elaborated them. He produced suppuration in the injured bones of animals after having injected into their veins putrid material or cultures of lactic ferments, consequently he attributed small value to Becker's results. He made cultivations of the same cocci which others had studied and, with Ogston, gave to them the names by which we now know them. It was in 1881 that he cultivated the staphylococcus from the pus of a case of osteomyelitis. In one case he found both the albus and aureus combined;

in another the albus alone; and in a third the aureus and streptococcus were found together. He produced the same result by injecting a pure culture of pyogenic microbes from a furuncle of the lip, as Struck did with cultivations from osteomyelitic pus.

Krause, in 1884, cultivated from osteomyelitic pus staphylococcus aureus and albus, which he found also in the effusion of the joints, occurring as a complication of this disease. He stated that when a bone was fractured subcutaneously, after the injection, he usually observed suppuration at the seat of the fracture, and from this pus the staphylococcus could again be cultivated. He endeavored further to produce a malady analogous to osteomyelitis without effecting such osseous injuries upon the animals. He was forced to the conclusion that there exists a coccus, in cases of acute infectious osteomyelitis, of extraordinary pathogenic and pyogenic powers, which, introduced in sufficient quantities, produces in animals acute infectious disease that leads inevitably to death, and which localizes itself by preference in the organs of locomotion, muscles, joints, and bones. His investigations were corroborated by Garré, who in addition showed that the staphylococcus is also present in such cases in the blood.

Ganglophe and Rodet, experimenting in the same direction, came to nearly the same conclusions, agreeing in the description of the microbes especially at fault; and these are now everywhere recognized by their biological characteristics, and everywhere described under the same names. Until recently, opinions have differed as to whether there was a specific germ of acute osteomyelitis alone, which did not manifest pathological properties in other directions, or whether this was a condition which might be called out according to circumstances by various forms. With regard to this question, we shall have more to say a little later.

Rosenbach selected the bone marrow for his experiments, principally for the reason that it is most completely protected from external influences, such as the penetration of exciting inflammatory agents from the skin, or, in fact, from any direction save the circulation. He demonstrated that neither mechanical injuries, comminution nor destruction of the marrow, nor such physical influences as the cautery or the electrical current, nor the action of chemical caustics, could produce a suppurative phlegmon in the bone marrow. He demonstrated further, on the contrary, that this medullary matter was extremely sensitive to the influence of infectious materials, or to rancid butter or products of decomposition, any of which speedily evoked violent symptoms of septic inflammation. Krause made the further valuable observation that all the common

changes noticed in muscles, kidneys, etc., which are met with in pyæmia are found as well in cases of acute osteomyelitis. He observed only those changes at the spot of his experimental fractures which were called forth or met with in cases of ordinary septic poisoning, with this difference, perhaps—that the marrow at the site of the experimental fracture was more extensively involved than after ordinary injection of septic material.

Pathological Anatomy.—A most important fact was comparatively early established, largely by the French writers, that in acute osteomyelitis the disturbance begins in the neighborhood of the epiphyseal cartilage, and that by early operation (trephining) the presence of pus can be demonstrated in the interior of the bone before it can be found anywhere else.

In the Memoirs of the Society of Surgery, 1855, Gosselin expressed himself as follows:

"When a bone is seized with diffuse ostitis the marrow participates in the inflammation. This marrow becomes highly infiltrated with blood and its vessels congested, plastic material is exuded from these same vessels and a portion of its fatty and albuminoid tissue is mixed with blood . . . Such a mixture of hydrocarbons and albuminoids is certainly one whose putrefaction would most rapidly lead to putrid poisoning, and whose putrid products are situated in a peculiarly favorable position for distribution throughout the system."

Some of the French authors, in describing the appearance of the interior of the bone, in serious cases of this lesion, have spoken of the medulla as exhaling an odor of infection. Some, like Reynaud, have supposed this to be due to gangrene of the same; others, like Gosselin, believe it due to putrefaction of the albuminoid and fatty elements of the bone marrow. It would appear that both mean practically the same thing, and they dignify it with the term *osteomyelite putride*. Of course, this means, in effect, a mixed infection, the ordinary putrefactive bacteria having also found entrance; or it may be due to the presence of Rosenbach's bacillus pyogenes fœtidus or saprogenes.

The macroscopic structure of the parts whose diseases we are studying has no small importance in our present investigation. No one has given this matter more attention than Ollier, and I may be permitted to quote one or two of his observations:

"The connecting cartilage is intimately adherent to the periosteum, with which it is continuous and blended at its circumference. This continuity is well seen in separations of the epiphyses, when the extremity of a diaphysis, separated from its cartilage, breaks through the ensheath-

ing periosteum and protrudes through the tear in the midst of the muscles." (*Int. Enc. Surg.*, vi. 885.)

Suppurative periostitis is, strictly speaking, endoperiostitis, as Ollier has shown; that is, it is the deeper layer of the membrane which is mainly and principally involved, and the bone must, in consequence, suffer more or less. It may, moreover, be primary or consecutive.

It is probably the cases of endoperiostitis in which the presence of bacteria does not figure, which lead to the rare condition of *periostitis albuminosa*, where a ropy, transparent fluid is met with instead of pus. These are to be regarded as inflammations which have been preserved from bacterial infection. In one instance of this kind that I have met with, neither the microscope nor culture experiments revealed the presence of microbes:

"The activity of the subperiosteal elements explains how the internal surface of the periosteum is also frequently the point of departure for osseous inflammations, which may be limited or may extend. In the latter case they spread all around the bone, and may reach the medulla through the juxta-epiphyseal regions, bringing about those isolations of the diaphysis, or those more or less extensive necroses which so often necessitate our interference." (Ollier.)

Besides these well-known and now commonly-recognized forms of parasitic ostitis and periostitis, there is yet another rare one, which, in 1883, Kiener and Poulet described under the name of *ostéite tuberculeuse aiguë envahissante et suppurée;* a species of tubercular ostitis, usually central, of such acuteness that it deserves to be regarded as coming justly within the province of this paper. The greater part of the epiphysis is rapidly invaded and the inflammatory disturbance is most intense. On section, the interior resembles a section of a mass of ice-cream, part vanilla and part raspberry. Small abscesses, even as large as an almond, of yellowish tint, are found in the bone. In the whitish parts the bony partitions are hard, thickened, and the medulla infiltrated with caseous products. The reverse obtains in the reddened areas. If a sequestrum reaches as far as a joint, it perforates the articular cartilage and may fall into the joint cavity, determining there, of course, a purulent and fungous arthritis.

Some time since, I had to disarticulate at the shoulder-joint an arm in which a most typical picture of this rare form of acute disease was presented. At each end of each long bone in the extremity existed the peculiar lesions above alluded to, in their most illustrative form. This was before attention had been called to this peculiar form of disease,

and yet my record of the case at the time, and the bony specimens still in my possession, stamp it as a typical case.

Rodet was the first to produce osteomyelitis in animals without inflicting a trauma before or after the injection. This he did by practising intravenous injections. The purulent inflammation, which was usually circumscribed, was commonly located near an epiphysis; it seldom extended over a large portion of the shaft. In many cases diastasis occurred, and frequently arthritis of the adjacent joint. In the most acute cases the animal died within twenty-four hours, with scarcely any change in the bone. The detection of microbes in the blood was the most difficult. They were most easily found in the kidneys, in which multiple abscesses were often met with. Subcutaneous injection resulted only in local infection; osteomyelitis could be produced only by intravenous injection. Young animals were the most susceptible. As the result of his observations, he considers the medulla, in close proximity to the epiphyseal line, as the primary seat of osteomyelitis of long bones. When epiphyseal separation occurred the pathological fracture was always on the side of the diathesis.

The explanation of these results is not difficult. Cocci may be carried directly to the vicinity of growing bone-marrow by direct blood-current, or they may be lodged upon a mucous membrane and here cause a local inflammation with local stasis, and then, after causing adhesion of infected leucocytes and local thrombosis, they may be carried along and lodged in some vessel too small to permit their passage. Should accident determine this to be in the bone, then we have the conditions under consideration as the result.

Predisposing Causes.—In these cases as in every other acute illness, we find certain conditions of the system which we may consider as predisposing causes. Our most typical illustrations are met with in the adolescent, where, as is well known, there is a maximum of circulatory activity in the neighborhood of the epiphyses. It is not saying too much to claim that where we find the greatest nutritive activity we get our most disastrous results from sudden changes. Let these changes be produced by injury, even slight, or exposure or reflex influences, so that a certain stasis shall ensue, and we can then better imagine how, when once a comparatively small number of pyogenic cocci have gained entrance through the blood-channels, they rapidly multiply, causing coagulation-necrosis, and become agents for the most active destruction and evil. Another anatomical fact is of great importance in this connection; the bone marrow is especially predisposed, in composition as in histological structure, to collect and retain germs of disease. It is,

moreover, most sensitive in its reactions, and there is further to be considered its now well-known function in the perfect elaboration of the blood. When, therefore, it is attacked, not only is there local disease, but there may be readily brought about serious and rapid alterations in the blood and conditions of true toxæmia.

It is, furthermore, at the age of childhood, as Ollier puts it, "That the action of cold, forced exercise, fatigue of the skeleton, or juxta-epiphyseal strain, gives rise, under the influence of a scrofulous or rheumatic predisposition, or of a general systemic poisoning, to those acute or subacute lesions," at points, "where increase in length takes place, and under the periosteum, where increase in thickness occurs." Almost all authors have recognized a predominance of this disease in the male sex. According to Lannelongue, out of one hundred and twenty-two cases, eighty-six were in boys and thirty-six in girls. Many writers unite in mentioning scrofula as a predisposing cause, and this must result not only from the cachexia or diathetic condition which the term scrofula implies, but also from the fact that active scrofulous lesions are tubercular lesions, and that the breaking down of tubercles in bones is a condition of mixed infection, which is simply intensified in activity and abbreviated in time in our most acute cases.

Privation and unhygienic surroundings play also a conspicuous *rôle* as predisposing to this disease. Whether acute articular rheumatism may be included in this category, is a question not yet settled. When we consider how rare suppuration is in connection with purely rheumatic affections, we may tentatively, at least, leave it out from further consideration.

We are justified in laying great stress on fatigue and exhaustion as predisposing causes.

It is not alone disease nor, in fact, any absolute diseased condition which predisposes to osteomyelitis; bad hygienic surroundings, as will be seen among the children of the poor in crowded cities, long forced marches, even lack of proper rest or of proper food, play almost as large a *rôle* as do actual injuries. The statistics of wounds among the insurgents of the Commune are sufficiently eloquent in this respect.

I am convinced, with Ollier, that hitherto we have not ascribed enough importance to the matter of juxta-epiphyseal strains in delicate and scrofulous children, as an active factor in producing acute suppuration of the parts. These are produced by falls, or by violent movements, which may be easily overlooked or forgotten. Minute separations, trabecular fractures or hemorrhages are thus caused, which suffice,

together with the vulnerability of the system so well recognized in such children, to lead to extensive disaster.

He (Ollier) speaks of the extension or migration of the inflammatory disturbances alluded to from one end of the diaphysis to the other, along the periosteum or medullary canal, sometimes without stopping, and going on to suppuration at both extremities of the shaft, so that we have to deal with two distinct centres of suppuration; and he denominates this *bipolar ostitis.* This is especially common in the tibia, and, I doubt not, many of you, like myself, have seen repeated instances of it.

Some French writers have spoken of pericarditis as a predisposing cause. In the writer's estimation, this is more likely to have been either a coincident lesion or a consequence, since the same bacteria are involved in each case. The same may be said of endocarditis. The most active occasional causes are strain, exposure, or excessive fatigue, or all three combined, especially if occurring in a patient with a tuberculous taint or diathesis.

Inasmuch as the extremities usually suffer most from these causes, it will be seen how easily such circulatory disturbances as partial or complete stasis may be thereby brought about, and how many organisms circulating in the blood may most easily find here a suitable culture-medium, surrounded by fewest disturbing elements. The French surgeons, especially, discussing this topic have shown considerable acerbity as to whether the periosteum or the medulla is first involved. This seems to have been quite unnecessary, if we remember that the bone-marrow extends in effect to the very surface of the bone. After all, it is a matter of very trifling importance; inflammation begins at either point, extends rapidly to the other, and, in our worst cases, we have to deal practically with a phlegmonous peri-osteomyelitis.

Osteomyelitis rarely affects short or flat bones; one meets with very few such cases. There is during the growth of long bones a concentration of nutritive activity, which within certain limits is intense; a concentration made necessary by the necessity for their growth mainly in one direction—that is, length. This growth of long bones in one direction is characterized by a great vascular richness, by a most active proliferation, by much energy in young tissues which are quite delicate, and which are subject to invasion by infectious agents. This may figure as the sole factor in producing acute osteomyelitis, and, though these infectious agents have the power of determining an explosion of disease, as it were, still we cannot be blind to the fact that their activity is prompted alone by the alterations which are so rapidly taking place in the ends of the young bones. The short and flat bones, as well by their

situation as by their function, are much less exposed to these alterations; their centres of ossification are not as localized, while ossificatory activity is much milder and less susceptible of deviation.

We are forced to recognize, moreover, cases of acute inflammatory disturbances upon the articular side of the epiphyseal line. In this case care will be required to discriminate between an acute suppurative synovitis and an acute infectious epiphysitis. Inasmuch as intense pain and tenderness, with muscular spasms, will generally predominate early in the latter case, we shall not have much difficulty in this respect. Epiphyseal separations have occurred in cases where this disaster has been veiled in the general features of an acute pyarthrosis.

In a recent thesis Lemoyne has summed up certain conclusions which may be appropriately reproduced here:

"First, that acute suppurative inflammation is frequently met with; second, that it is manifested in two forms, either as phlegmonous periostitis more rarely, or as acute osteomyelitis more commonly; third, that the disease is of an infectious nature, the causes formerly evoked not being sufficient to explain its development; fourth, that the symptoms in the adult are quite analogous to those in children; fifth, that when inflammation is limited to the deeper layers of periosteum recovery is more likely without necrosis or exfoliation; sixth, that the reparative process is often followed by more or less hyperostosis; seventh, that the prognosis is more grave after the individual has reached the twenty-fifth year of life."

As illustrating the rapidity with which destructive processes may take place, let me quote the following statement by Ollier, made at the first Congress of French Surgeons:

"Rodet has been able to reproduce all the varieties of juxta-epiphyseal ostitis by inoculations with fluid cultures of the Staphylococcus aureus. I have had opportunity to examine some of the specimens thus obtained, and in some of them have been able to demonstrate the presence of a sequestrum even as early as two or three days after inoculation."

The usual form of lesion observed by Rodet was rarefying; exceptionally it was condensing.

A case reported by Gerster shows that extensive necrosis may take place in three days after the inception of the disease. He believes, as I do, that a few hours are sufficient to determine its occurrence. Perhaps the most rapidly fatal case on record is that reported by Kohts, in which the disease began and ended apparently in thirty-six hours. (Right femur involved.) The reporter regarded the peri-bronchial tissue as the

port of entry in this case. Most painstaking investigation revealed fat-embolism of the lungs, pulmonary infarcts, purulent peri-bronchitis, "myositis mikrococcica"; bacteria were found, as well, in the capillaries of the spleen and kidneys. The peri-bronchitis in this instance was ascribed to a tumble into the water which had befallen the child three weeks previously.

The complications of acute infectious disease in bone are mainly in the direction of septicæmia and pyæmia, except so far as local destruction is concerned. If circumstances permit a reasonably early escape of pus, the final septic disaster may be averted. When this does not occur, and when the surgeon fails to effect an escape of pus and relief of tension, a patient is pretty sure to die of pyæmia. If we are right in speaking of these cases as in the first place a local pyæmia of bone, we shall more readily understand how easily and naturally this result is brought about. As Verneuil has put it:

"Lesions of bone are predisposed especially to severe septicæmia. They prompt and favor continuation and prolongation of dosage of putrid poisoning. The poisonous material is shut in in such a way that escape by way of the vessels and final circulation is almost impossible."

Accepting this view, we can understand both the justness of P. Wagner's expression *kryptogenetische Pyämie* as furnishing a clew to the concealed origin of some previously inexplicable cases of idiopathic pyæmia, and of Leube's *spontané Septiko-pyämie.*

Indeed, the view that acute osteomyelitis sustains a close relation to pyæmia is hardly expressing it strongly enough. It not only leads to pyæmia, but it is, almost from the beginning, a pyæmia. This is to be explained, of course, on the anatomical ground that osteomyelitis is essentially a septic phlebitis of bone-marrow, and when we remember the most important *rôle* played by the veins in the production of the pyæmic condition, we can see how almost immediately an osteomyelitis, regarded in this light, becomes first a local and then a general pyæmic condition.

There is No Specific Germ of the Disease.—Osteomyelitis is, in the light of these and other facts to be adduced, not to be regarded as a specific disease, but as one manifestation of the pathogenic properties of several microörganisms possessing common specific pyogenic activities. It is a phlegmon of bone, or a local pyæmia.

Experiments to ascertain the extent of diffusion of an osteomyelitic microbe have been made by Ribbert. Twenty-four hours after direct injection he found them in all the organs; but later, only in the kidneys. In regard to their localization, three conditions must be taken into con-

sideration: *First*, embolic obstruction of capillaries; *Second*, elimination of pyogenic cocci through the kidneys; *Third*, influence of traumatism. Lübbert entered into a most careful study of the alleged coccus of osteomyelitis, and established for himself its complete identity with the Staphylococcus aureus. His monograph also demonstrates the diversity of the lesions caused by this organism. He further showed that *en masse* it was more resistant to elevated temperatures when dry than when suspended in water. When dry it withstood a heat of 80° C. (176° F.) for nearly an hour. Like many other pathogenic microbes, the more it is cultivated in living animals, the more intense, apparently, becomes its activity.

From his biological study, Lübbert was led to the conclusion that the intensity of action of the Staphylococcus aureus varied greatly without sufficient cause of variation being known. Inoculation of granulation surfaces proved harmless; even on superficial abrasions it seemed to produce no effect. Subcutaneous injections resulted in the formation of abscesses of varying degrees of severity. Injections into the pleural and peritoneal cavities were oftenest followed by intense symptoms; intratracheal injections produced suppurative tracheitis and foci in the lungs; intravascular injections were followed by septic symptoms, with foci in the intestinal mucosa. Feeding experiments proved harmless. Why the staphylococcus forms should at one time produce a superficial abscess, at another a deep and disastrous phlegmon of bone, and at yet others an endocarditis, a pericarditis, a brain abscess, a purulent synovitis, or any one of many other manifestations of its presence, we can, as yet, only partially answer. When we can formulate an accurate response to this query we can tell, also, why rarely a subcutaneous fracture suppurates, or why, as in an instance related by Bergmann, a piece of cloth encapsulated in the deeper tissues for fifteen years should finally set up a typical abscess.

Bertoye came to the conclusion that the S. albus was less malignant in its action in bones than the S. aureus. He thought, moreover, that S. albus, after a series of cultivations through living animals, gradually became converted into the other variety, *i. e.*, aureus.

Garré's experiment of applying a quantity of pure culture of S. aureus taken from a case of osteomyelitis to the unbroken skin of his own arm, and thereby producing a typical carbuncle, shows how it is not always necessary to invoke the hypothesis of a precedent lesion in order to explain the occurrence of a deep-seated phlegmon.

Kraske has also had an opportunity to observe a case illustrating the

same identity between the microbes met with in osteomyelitis and carbuncle.

The writer's own studies of a few carbuncles have shown him that the Staphylococcus aureus is almost always to be met with in this affection, along with other cocci. Out of five cases of osteomyelitis bacteriologically studied by Kraske, the Staphylococcus aureus was twice found alone; in the other cases other species were also met with; some of these forms being present probably by accident, and some of them presenting forms not yet identified, whose consequent pyogenic importance is not yet recognized. Kraske takes the ground that the somewhat various features and courses pursued by different cases of osteomyelitis may be, in some degree, at least, owing not only to the presence of the various pyogenic cocci or to their contamination with other forms, but also to the various ways in which infection may have occurred.

A complete recognition of the absolute etiological identity of acute spontaneous and acute traumatic osteomyelitis can only be insisted upon after extensive bacteriological investigations. It may make a difference as to whether the microbe is introduced directly into the bone, as in injury, or by bloodvessels, as in the idiopathic forms. The character of the inflammation may also vary according to the culture-medium in which the germ lodges, just as it makes a difference in laboratory experiments whether one makes a needle or a plate culture.

Kraske considers that recurring attacks are not necessarily an indication of former infection, but may arise entirely *de novo*. The results of his clinical studies are, first, the Staphylococcus aureus can produce osteomyelitis, and as in fact most frequently met with in osteomyelitic products; second, in a certain number of cases acute osteomyelitis is the result of a mixed infection, and is then most prone to produce a severe case. He thinks that the localization of cocci in the neighborhood of epiphyseal lines in young persons is due to the peculiar location and arrangement of the capillary vessels in this neighborhood, and that, almost without exception, the inflammatory process has its origin here; that the rapid local spreading of the disturbances is in large measure due to the unyielding nature of the tissues around the primary trouble, and to the fact that the bloodvessels act as direct disseminators, their contents forming the nutrient material for the microbes. Thrombosis is a coincident and necessarily very early condition in every case of acute osteomyelitis. When the microbes reënter the unobstructed vascular channels again, we generally get dissemination and metastatic foci, with coagulation-necrosis of the intima and thrombosis locally. As is well known, in some cases, even of external infection, early in the disease the

constitutional symptoms are entirely disproportionate to the local lesions, giving us a clinical picture of intense septic intoxication. This is in all probability an acute ptomaïne-poisoning, which may possibly be due in its rapidity and intensity to the character of pabulum furnished by medullary tissue.

From some of my own cases that I have tried to investigate carefully I can report the following findings:

I. Case of acute lumbar disease, running a rapid course and terminating in abscess and caries. Found only the Staphylococcus pyogenes citreus.

II. Case of acute central ostitis of humerus. Found Staphylococcus pyogenes albus and aureus.

III. Case of acute necrosis of tibia, following osteoperiostitis. Found Staphylococcus pyogenes aureus and cereus flavus.

IV. Case of bone abscess of the femur following acute juxta-epiphyseal osteomyelitis. Found Staphylococcus pyogenes aureus and cereus flavus.

V. Case of acute central osteomyelitis of clavicle, resulting in nearly total necrosis. Found Staphylococcus pyogenes albus after cultivation, and tubercle bacilli in the pus, *i. e.*, mixed infection. This case (child) I had operated upon before, having removed the entire lower jaw (a half on each of two occasions) for trouble of similar character.

VI. Case of acute and rapidly fatal osteomyelitis of femur in a young girl. Amputation at hip-joint in desperate attempt to save life. Found Staphylococcus pyogenes aureus and Streptococcus pyogenes.

VII. Case of acute osteomyelitis of tarsus, with extensive destruction of the same. Found Staphylococcus pyogenes aureus.

It falls to my lot to see many cases similar to those above reported, but circumstances have either prevented any examination of the others or my results have seemed not quite worthy of reliance.

The view that each of these conditions is due to a distinct specific form is now well-nigh abandoned. For my own part, I have, in a case of puerperal septicæmia, accompanied by ulcerative endocarditis and multiple but apparently not metastatic abscesses, many of them subperiosteal, recognized the Staphylococcus albus by careful culture-experiments; consequently I record it as but another expression of specific infection in connection with acute deep phlegmon. We meet also with acute endocarditis which may or may not be fatal. Just such cases have not presented in my own experience, but one may find them recorded, as, for instance, in the thesis of Benoit (Paris, 1876, No. 308).

It is now generally agreed among pathologists that the staphylococci

are the causative agents not only in pus-production, and its various manifestations as seen in the periosteum and the bone, but also in the production of acute ulcerative endocarditis, as well as of most of the other suppurative disturbances met with in these cases. Furthermore, that they, probably, with the streptococci, are at fault when we have to deal with septic infection, whether it appears as sapræmia, septicæmia or pyæmia, and whether it occur apparently spontaneously, or in the puerperal state, or in any other form. I think we may say that we do not at present know a form of this septic infection which can be separated from the presence of these organisms.

Staphylococcus Re-infection.—There seems to be no denying the fact that collections of pyogenic and pathogenic organisms can shut themselves off in certain parts of the system where, as isolated cells of low formation, they remain quiet for months or years until called into activity by some new or special provocation. It is not overstating the matter to claim that isolated collections of pyogenic cocci remain quiescent for long periods of time, especially in the bones, even for twenty years, or longer, then to break out again, as it were, into a conflagration, when anything happens that may permit the outbreak; prolonged exposure, dissipation, or overexertion may bring this about; so may such wasting diseases as typhoid; so may also pregnancy, the puerperal state, puerperal fever, or any other local infection, and so may also such injury as a fracture. There is nothing stranger in all this than it is strange that grains of corn or wheat may be kept for decades or centuries and then, under favorable auspices, once more resume cell-activity and cell-proliferation. This statement finds many clinical illustrations, as when we have occasion to remove sequestra from limbs which show many old scars of old bone abscesses, or when we even have to amputate for destruction too extensive to permit milder measures.

In fact, in most cases of osteomyelitis in the adult we shall find, as Lannelongue has told us, vestiges of similar trouble dating back to childhood or infancy.

I have recently had in my own practice a case where I had to amputate a thigh for acute necrosis, resulting from acute osteomyelitis, where the last outbreak of bone trouble of which I could get any account dated nearly twenty years previous. I feel as confident with this case as I do with anything which I cannot demonstrate, that a collection of microbes of years' standing had been excited into activity as the result of overexertion and prostration. So, too, Volkmann says:

"I have already observed five or six cases where patients who had suffered from acute periostitis or osteomyelitis, and who exhibited ex-

tensive bone scars, have been suddenly seized after ten, fifteen, even twenty years, with acute diffuse inflammation which proceeded from their previous disease, and exhibited a peculiarly severe destructive character. Four of these patients died; two of them acute pyæmia; in one case amputation of the thigh was necessary."

Gerster has reported a case of apparent quiescence of forty-seven years between the attacks, the disease recurring after this period at the same spot.

Rosenbach emphasizes the comparatively long period of time during which cultures remain active, and calls attention to the clinical manifestations of this fact in that suppuration may be resumed in bones, at points where previous lesions had existed, long after the original disease had apparently subsided.

Were it worth while, I could give many other illustrations of this clinical feature from my own practice, including several in which limbs have had to be removed to secure relief.

Kraske makes the important point that by no means every case of recurring osteomyelitis is really such; quite a large proportion of them depend upon a new infection, determined by certain changes in the bone-marrow, which later, at an age when the disease is most common (period of youth), predispose to fresh inoculation. The rest of these cases undoubtedly are connected with the long period of quiescence to which I have already alluded.

Ports of Entry.—Observers have long noted the coincidence of osteomyelitis with various excoriations, abrasions, wounds, and superficial or deep lesions, but without drawing, until lately, the conclusion that the osteal lesion was the result of infection through these channels. Chassaignac was the first to trace a causative relation, as he did in the case of a boy whose ankles had been chafed by his boots and who had a very severe osteomyelitis of the right femur. Several of the earlier observers in this direction have remarked their cases occurring in those who have recent or older scars. Camps has called attention to the fact that in cases involving the phalanges small lesions about the fingers have usually preceded. And so osteomyelitis of the lower jaw frequently follows some local affection, usually from inside the mouth.

A point of the greatest importance, therefore, is to recognize, if possible, the path of infection. As we understand it to-day, infection may occur through the skin, through the respiratory and circulatory organs, through the mucous membrane or intestinal canal, and through injury by direct penetration.

It is more than possible that the source of infection in acute osteo-

myelitis may be a suppurative focus upon or just beneath the skin, and that this, even, may be so minute as to be unrecognizable. A furuncle of the lip has been known to be the exciting factor in such cases as this.

In fact, Kraske is of the opinion that in the majority of cases the infection proceeds from the skin or the subcutaneous areolar tissues. Kocher had already called attention to his so-called secondary osteomyelitis, and the fact that in seeking for its causes one must not forget to examine carefully the entire surface of the body, and next to this the lymph glands, just as in a large portion of the formerly so-called "scrofulous glands" one could discover almost always some epithelial defect through which infection could take place, and from which swelling and suppuration of these glands ensued.

It is important in this connection not to overlook the tonsils as the possible seat of infection, since more than one observer has found their tissues more or less loaded with the Staphylococcus aureus, and Kraske considers it quite possible that a general infection may follow the growth of staphylococci in the glands.

The recent thesis of Ayala-Rios contains some carefully-reported cases showing how osteomyelitis has followed, in one case, chilblains; in a second, a felon; in a third, chafing and abrasion of the scrotum and groin; in a fourth, abscess; injury to head in a fifth; chafing and lymphangitis in a sixth; and furuncle, aphthous ulceration of the mouth, and other ordinarily mild and superficial bruises or lesions in yet others. In seven (*i. e.*, one-half) of his cases the serious osseous disturbance seemed to be in close relation—in point of locality—with the point of entrance of the microbes; in the other seven this close relation did not obtain.

Gerster has reported a case of extensive rapid destruction of one ischium with extensive purulent infiltration, the patient having had chronic suppuration in the middle ear.

Kraske asks the question, "Is it possible or probable that osteomyelitic infection may result from the alimentary tract?" Kocher has been decidedly of the view that this is possible. The infrequency of such infection is, of course, only negative. We know that both anthrax and tuberculosis can be produced by the ingestion of infected food. The peculiarity of Kocher's experiments may, perhaps, prevent us from completely accepting his views, and yet we cannot contradict the evidence of his two cases, in which he seems to show clearly that the intestines were the source of infection. We are in position, then, rather to say that the intestine is a possible source of infection than that it is a

well-known source. Much more probable is infection through the lungs, and, consequently, the circulation. We are as well aware, of course, of the transfer of organic cells as of inorganic material from the respiratory organs to the bronchial glands; this being the fact, the general or more distant local infection is generally a difference in degree.

In a case of acute osteomyelitis two paths of exit are open for the pus: one by way of the joint, the other through the periosteum—the latter being the more frequent. It is, in fact, cases of the latter variety, where the pus forms in the middle of the long bones, which have led to the error of mistaking many of these for cases of simple acute suppurative periostitis. Such a case will be presented to a surgeon for the relief of what is supposed to be a simple subperiosteal abscess with, perhaps, superficial caries, but which will be found to be a case of deep central lesion. In the past few weeks a young lad was brought to my clinic whose case had been regarded as simply one of acute periostitis. I learned that he had suddenly been seized, in September of last year, with severe pain in one leg, which rapidly became intense. His case was for two or three weeks a very serious one; his fever was high, he was delirious much of the time, and even his life was despaired of. Later fluctuation was detected, a small skin opening was made, and pus evacuated. After this his most urgent symptoms subsided. When brought to me, the latter part of March, I found four sinuses, near the middle of the tibia, discharging small quantities of pus; upon operation it was found that nearly total central necrosis had taken place, and it was necessary to remove, subperiosteally, nearly all the bone from one epiphysis to the other. Surely, no ordinary case of periostitis alone could bring about such deep and central destruction of bone as this case presented.

This was an an illustration of perforation of pus through the periosteum; on the other hand, we find either epiphyseal perforation with formation of extra-articular abscesses, or direct perforation of pus into the joint, with consequent necrosis and destruction of more or less of the joint-structures, and permanent loss of function providing that life be spared. The discovery of pus in a joint-cavity, in such cases, is not necessarily a sign that it has perforated from the medullary cavity. It may be empyema of the joint consequent upon the presence of pus in the neighborhood as we find ordinary empyema after acute specific process in the lungs proper, or it may be an expression of a metatastic process, in which case other manifestations of the same will probably be detected.

Mixed Infection.—We have two forms of mixed infection complicating acute suppurative changes in bone and periosteum, which certainly deserve brief special consideration. These comprise the typhoid and the

tubercular. Whether the syphilitic properly may form a third we cannot yet say. The tubercular form I have already alluded to when speaking of the clinical manifestations described by Kiener and Poulet. Not less interesting are the acute disturbances in bone consecutive to typhoid.

Ponfick's researches concerning sympathetic affections of bone-marrow after internal disease constitute most interesting reading, and are well worthy of study in this connection. They show how a large number of infectious diseases such as typhus, typhoid, intermitent fever, continued fevers, and the like, determine changes in the bone-marrow which are anatomically recognizable. This being the case, we can easily see how slight a provocation in the way of septic infection is necessary to set up acute and disastrous suppurative changes.

Typhoid bacilli have been found at wide distances from the intestinal canal; Gaffky has found them in the liver; Bouchard has met with them in the urine and in the kidneys. They have been found in hyperæmic lungs along with other bacterial forms. They have also been seen in connection with other lesions in furuncles, in bedsores, and in connection with other septicæmic lesions.

Whether the specific bacillus of typhoid is properly to be ranked with the true pyogenic bacteria, or whether its lodgement and the changes incident thereto simply predispose to a mixed infection with the staphylococcus or streptococcus, is a question, in my estimation, hardly yet to be positively answered. I do not forget that, for instance, Fränkel in 1885 carefully studied a case of post-typhoid abscess of the abdominal wall, from the pus from which he cultivated typhoid bacilli, and these only. His case certainly demonstrated that the typhoid microbe could remain in the body longer than was ordinarily supposed (four months in this case), and it would seem to show that rarely it can manifest true pyogenic properties; still, such an investigation needs confirmatory evidence.

Of no small interest in this connection is a recent paper by Ebermaier (*Archiv für klinische Medicin*, Bd. 44, Heft 2.) In two cases of periostitis, occurring as a complication of typhoid fever, he was able to prove the presence of the typhoid bacilli in the pus and blood present. The first case was in an eighteen-year old lad, in whom, on the thirteenth day of the disease, signs of a periostitis of the second metatarsal bone of the left foot appeared. On the twenty-first day fluctuation was present, and the abscess was opened. Examination of the pus showed the presence of a few typhoid bacilli, and cultivation confirmed the microscopic result. The second case was in a nineteen-year-old seamstress, in whom a peri-

ostitis of the right tibia developed during convalescence. The affected spot was incised; no pus obtained, but much blood. Cultivations obtained from this gave the typhoid bacilli. In neither were any other micro-organisms found. He gives six other cases of periostitis occurring during the course of typhoid fever, one of which went on to suppuration; he thinks that the results obtained from the first two cases allow us to consider the typhoid bacilli as their causative agent. He considers that the bacilli reach the periosteum from the medullary part of the bone through the Haversian canals, and is led to this belief from having obtained cultures of the typhoid bacilli from the medullary part of a rib removed post-mortem from a fatal case of typhoid fever. Whether this affords all the evidence needed each one of us must determine for himself.

At all events, I can state of my own knowledge, not all post-typhoid abscesses contain such pure cultures of typhoid bacilli, since in at least one such case I have been able to cultivate the common Staphylococcus aureus from the pus, along with the bacillus in question.

Post-scarlatinal abscesses are certainly usually mixed infections, providing the alleged germ of scarlatina has aught to do with them, since in cases of this kind, too, I have found nothing but the common pyogenic forms, even after careful plating. When we know positively what microörganism it is that produces scarlatina, we can speak with more certainty about mixed infection in this connection. So, too, with the other exanthemata. Rarely we hear of cases of acute bone trouble following after variola.

In the *Archives of Medicine* for 1830 (vol. iv. page 491) there was reported by Ancell a case of a babe of eleven months suffering with smallpox. An eruption appeared, discoloration commenced on the tenth day; upon the eleventh day the infant showed signs of distress; various swellings occurred in the neighborhood of the joints; fluctuation soon followed, with dilatation of superficial veins; fresh swellings appeared, crepitation was noticed in the joints and on pressing the fingers; even the costo-vertebral joints presented crepitant tumors. After death diastasis of most all of the epiphyses was noted, while the various joints were filled with pus.

In closing, let us now see to what extent we can answer the questions propounded at the beginning of this paper. I think we shall be perfectly safe in adopting the following conclusions:

I. There is no one specific microbe for the production of acute infectious processes in bone.

II. Most, if not all, of the staphylococci can cause them. Exception-

ally, the streptococci may exert such an influence. So may tubercle bacilli and those of typhoid.

III. Commonly, when these latter are met with, we have to deal with a mixed infection, the pyogenic cocci being the final and active destructive agents.

IV. Of all forms, the Staphylococcus aureus is the most pernicious.

V. The parasitic infection may occur through the ears, eyes, nose, mouth, pharynx, respiratory passages, mucosa of the alimentary canal, or skin; or, in other words, through any lesion of the external or internal body surfaces. Furthermore, from any subcutaneous phlegmon, however small.

VI. The infection need not necessarily have been recent. The staphylococci and the bacilli of tubercle have the property of hibernation, so to speak, and their period of latency is one of almost indefinite. length; this is especially true of the former.

VII. Some predisposing condition will be usually found to have favored the entrance of the infectious germs into the system and their lodgement in the localities where they manifest most activity.

VIII. Of the general causes which favor their entrance the diathetic conditions, such as the tubercular, and the dyscrasial, like the syphilitic, also the post-febrile, play a most conspicuous part. Age, *i. e.*, childhood, is also always a predisposing cause.

IX. Of the causes which favor lodgement in particular localities, exposure, exhaustion, strain, and more marked traumatisms are those usually met with.

X. Pathologically, a case of acute infectious osteomyelitis becomes within a few hours a case of localized pyæmia, with all that the term implies; and this within a few hours more may become so generalized that a patient may die, even within thirty-six hours after noting the first symptom, of overwhelming septic intoxication.

XI. Acute infectious periostitis may run almost as acute a course, locally, as the same grade of osteomyelitis; but, inasmuch as pus finds more readily an outlet, it seldom leads to as rapid general disaster.

XII. A final conclusion in a therapeutic direction may, perhaps, be permitted here. No matter how former surgeons, like Demme and Chassaignac, disputed the matter, there is to-day but *one* safe and rational treatment, and that is, early and radical operation. These cases have been too often considered to be acute rheumatic affections, and many lives and limbs have been sacrificed to this diagnostic error. Our books and teachers need to give more exact instruction in this respect. When students are taught to recognize early the gravity of these condi-

tious, they will learn that which, in many schools at least, they are not taught to-day. And now that we no longer dread as we did a combat with these insidious and invisible foes, there is no longer any reason for hesitating to operate early, since it offers our sole and almost only hope. Early, radical, and antiseptic attack constitutes, then, the therapy of the diseases to which I have invited your attention.

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April 25, 1889.

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11. *Excisions of knee-joint.*

Exhibited by Dr. H. R. Wharton.

1. A specimen from a case of excision of the knee-joint removed from a man, a patient in the University Hospital, by Prof. Ashhurst.

The patient from whom these specimens were removed slipped and fell, injuring the left knee, in October, 1887. He was laid up after the accident, and has not been able to use the knee since. A short time before his admission to the hospital the knee became swollen and painful, and when he was admitted to the hospital the left knee was moderately flexed and the tissues in the neighborhood of the knee were greatly swollen and painful. The patella was floating, and there was marked lateral motion of the knee-joint.

Considering the age of the patient, amputation was suggested as the safer procedure, but as the patient was unwilling to submit to this operation, and as excision of the diseased joint upon exploration was found to be a favorable operation, it was decided upon.

The bursæ adjacent to the joint were found filled with purulent fluid,

and also the knee-joint; the articular surfaces of the bones were found in a condition of acute ostitis, with exfoliation at numerous points of the articular cartilages. The specimens are of interest, as they show this condition very clearly, and it is not often that we have an opportunity of seeing the specimens in a case which has run so rapid a course.

2. A specimen of disease of the knee-joint removed from a patient in the Children's Hospital by amputation of the thigh.

The patient, Leo G., æt. 5 years, was admitted to the hospital November, 1887, suffering from caries of the spine and advanced strumous arthritis of the left knee-joint of more than two years' standing. Prof. Ashhurst, under whose care the case was, taking into account the patient's constitutional condition, considered it advisable to amputate the thigh rather than to attempt the more conservative operation of excision of the joint. This was accordingly done, and the patient made a good recovery. The specimen of the diseased joint I show this evening, and I have made a longitudinal section of the diseased structures which shows very well the condition of the diseased joint and subjacent tissues.

3. A specimen from a case of reëxcision of the knee-joint, with the following history:

Irma H., æt. 7 years, was admitted to the Children's Hospital in 1886 with arthritis of the left knee. At the time of her admission there were several sinuses discharging in the neighborhood of the knee-joint.

In December, 1886, I excised the knee-joint, and the patient did well. The wound healed promptly, and the patient's general condition was much improved. About six months after the operation the patient began to complain of pain in the excised knee, and several abscesses formed and were evacuated. At this time it was observed that the union which had taken place between the ends of the excised bones was softening down, and a certain amount of motion could be obtained between the ends of the bones.

The condition of the part not improving, on November 30, 1887, a reëxcision of the joint was performed, and the ends of the bones were found held together by a poorly-developed fibrous tissue, and the greater portion of the surface of the bones was found to be carious.

I am glad to be able to present these specimens, as it is not often we have the opportunity of presenting the specimens from a case of re-excision of a joint. *February* 23, 1889.

12. *Sarcoma of the tibia.*

Exhibited by Dr. H. R. Wharton.

A woman, æt. 23 years, was admitted to Prof. Ashhurst's ward in the University Hospital, November 20, 1888, who had been complaining for a short time of slight swelling and pain over the head of the right tibia. The case was supposed to be one of periostitis, and was put upon appropriate treatment. In spite of treatment, within a month the swelling had increased markedly in size and the pain still continued. It was now decided that the growth was a sarcoma involving the head of the tibia, and amputation of the thigh was recommended. The growth was removed by amputation of the thigh, and on examination it proved to be a periosteal sarcoma.

The chief points of clinical interest in this case are the symptoms which first attracted attention, resembling an attack of periostitis of the head of the tibia, and the subsequent extremely rapid development of the growth. *March* 14, 1889.

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13. *Central sarcoma involving the tibia.*

Exhibited by Dr. H. R. Wharton.

This specimen was removed from a patient in the University Hospital by Prof. Esmarch, by amputation of the femur in the lower third. The patient from whom this growth was taken was a woman, æt. 21 years, who first noticed a swelling below the knee of the right leg nine months before her admission to the hospital. The patient was pregnant at the time, and the tumor increased rapidly in size, and when I saw her, the day previous to her confinement, the tumor was larger than a man's head. Three weeks after her confinement the growth was removed by amputation of the thigh, and the patient has done well since the operation.

The growth appears to have originated in the head of the tibia, and is a central sarcoma. There was no involvement of the lower end of the femur, nor were the inguinal glands enlarged.

The speaker called attention to the more favorable prognosis which obtains in cases of sarcomata involving bone which are central in their origin, as compared with those which have a periosteal origin.

March 14, 1889.

II. THE DIGESTIVE APPARATUS.

1. *Cancer of the Stomach.*

Exhibited by Dr. J. H. Musser.

Absence of tumor or hemorrhage; short duration of disease; death following hemorrhage from the kidney.—J. P., æt. 46 years, married. German, glassblower, good family history, habits fair, although occasionally drank beer to excess; father of a large healthy family, subject to considerable care the past year. No previous disease and no history of syphilis. July 29, 1887, observed vomiting occurring after meals. The vomiting was not constant, and took place almost immediately after taking food. For some months previous was subject to pain and distress behind the sternum. He felt as if the food lodged just back of the middle of the breast-bone. This subjective sensation increased and was associated with much burning. After he began to vomit the sense of obstruction would be relieved. In June, 1887, he weighed 180 pounds; in October, when first under observation, he weighed 124 pounds. In addition to loss of flesh he was pale, short of breath on exertion, and complained especially of difficulty in swallowing. The obstruction appeared then to be back of the larynx and at the end of the breast-bone. The passage of a bougie revealed constriction at each place mentioned, five and one-half and sixteen inches respectively, from the teeth. Constipation was marked, hemorrhage had never taken place, the food vomited was not digested, and was not acid. On examination of the abdomen no tumor could be detected, but some slight tenderness on pressure only, was found. The abdomen was first full, but later became scaphoid. Daily dilatation of the apparent stricture was practised and the vomiting was relieved. It had continued from July to the middle of October. The patient gained somewhat in weight, but its figure varied from time to time. In consultation it was thought that stricture of the œsophagus of specific origin was the cause of the patient's ailment. The reporter always thought cancer was present. The middle of February his daughter's death caused extreme prostration, from which he did not rally. On the 28th of the month he had an enormous discharge of blood from the bladder, which led to greater

prostration, and was followed in two days by a stupor which ended in death on the 3d of March.

Post-mortem, twenty-four hours after death, limited to the abdomen. In the peritoneum covering the bladder, and in the region of the left iliac fossa, small, flat, pale nodules the size of the thumb-nail were seen in the peritoneum. Smaller cancerous masses invaded the peritoneum under the diaphragm of the right side. Otherwise the peritoneum was normal. One nodule the size of a half dollar about opposite the eighth rib in the axillary line, was particularly noted. It was adherent to the liver by recent adhesions.

On first opening the abdomen no tumor was seen; after displacement of the stomach, however, the mass hereafter described, was found at the point where the œsophagus passes through the diaphragm lying along the spinal column. The œsophagus was surrounded by the firm mass, so occluding its extremity that a lead-pencil could not be passed through the orifice. It invaded the walls of the stomach from this point irregularly for three or four inches distant from the cardia. Two glands back of the œsophagus were diseased, large, pale, and firm. The pancreas, spleen, and liver were free from disease, as also the lymphatic glands. Bladder was contracted, the mucous membrane showed recent hemorrhages underneath. The ureters were normal, the kidneys congested.

Remarks.—The absence of tumor during life, the absence of hemorrhage except a slight expectoration of blood four or five days before death, the rather rapid progress of the case, the improvement of the vomiting by the passage of a bougie, are worthy of note. The diagnosis was somewhat difficult, although the emaciation, the anæmia, the grave appearance of the patient, appeared to indicate a more serious disease than simple stricture of the œsophagus. A severe pain in the right axillary region over the surface of the liver appeared to the writer to confirm his notion of the presence of internal cancer, for to his mind he thought it due to a local peritonitis caused by a nodule in the liver, particularly as there were no signs of pleurisy. The liver, it may be said, was rather enlarged during life, and at the post-mortem was found of full size and fatty, the pain having been due to a nodule in the peritoneum covering the diaphragm. The improvement of the vomiting is of interest, and shows that the strictures were possibly spasmodic in character. It is true that, for some time and during the entire time under observation, the patient could not swallow large masses of food. Liquids, however, were also rejected prior to the dilatation. The autopsy showed the absence of stricture at the points previously noted,

and also the absence of dilatation of the gullet. Unlike several cases previously reported of cancer without tumor, there was not in this case any enlargement of the lymphatic glands above the clavicle.

November 10, 1887.

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2. *Fatty hypertrophic cirrhosis.*

Exhibited by Dr. E. T. Bruen.

The accompanying specimen of enlarged liver was obtained at the autopsy upon A. P., æt. 35 years, a native of Italy, who entered the Philadelphia Hospital September 29, 1887. When admitted to the wards the mental condition of the patient was dull, and but an imperfect previous history could be obtained. She stated that she had chills with fever for several months previously; that her skin had been yellow for ten days, and a cough had been troublesome for a week. The skin was deeply jaundiced, the abdomen prominent, so much so that pregnancy as far advanced as the seventh month was suggested. Physical examination disclosed a marked increase in the area of hepatic dulness. The outline of this organ could be readily detected in the right iliac region. Dulness on percussion and bronchial breathing were recognized over the lower lobe of the right lung.

Temperature record: 99.2° P.M. September 29th; 99.2° A. M., 102.2° P.M., October 1st; 101.3° A M., 100.2° P.M., October 2d; 100.2° A.M., 103.1° P.M., October 3d.

Urine was scanty, markedly colored by bile-pigment, and containing an abundant deposit of urates, and a trace of albumin. Cholesteræmia was marked. On October 1st the breathing was stertorous, and pupils contracted, with stupor. Death occurred October 3, 1887.

At the post-mortem the following notes were made: The body was that of a medium-sized woman. The skin was intensely jaundiced, the lemon hue less marked in the legs, very marked in the feet. The legs were also œdematous. Abdomen much distended, but no subcutaneous hemorrhages. On opening the abdomen a small amount of clear yellow serum was found in the cavity. Liver enlarged five inches below xiphoid, and in right nipple-line at least three inches below the margin of the ribs. Omentum loaded with fat, as were also the abdominal walls. Small intestines discolored.

Liver weighed 7½ pounds. It was intensely yellow, capsule with a

number of patches of old thickening. Gall-bladder contained small amount of bile; ducts pervious and normal. Structure of the liver firm and smooth, with general interlobular increase of connective tissue, grits on section. Small granular lobulation was noticed externally, and appearance on section was similar. Acini generally fatty, some, however, showing central vein with small area of brown liver-structure—the periphery in all lobules fatty. Mucous membrane of the stomach was the seat of intense hyperæmia, with submucous hemorrhages. Considerable amount of blood was found in the stomach. Duodenum hyperæmic.

Spleen, sixteen ounces in weight, soft, dark-red. In cæcum, extending along the course of the large bowel, inflammation with ulceration of the mucous membrane, also ulceration of solitary glands, and folds of a diphtheritic character—suprarenal capsules, pancreas, and retro-peritoneal glands normal. Heart weighed twelve ounces. Ante-mortem clots were found in cavities, muscles pale and soft, valves normal, coronary vessels normal, one or two plaques of atheroma in aorta. Bronchial glands normal. Both lungs adherent throughout by pleural adhesions with puckering at the apex of the right lung. The base of the left lung the seat of pneumonia in the first stage. Mucous membrane of bronchi in both lungs congested.

Kidneys enlarged, eight ounces each. The capsule was thickened and removed with difficulty, surface of kidney slightly granular, section granular, and cortex diminished; the appearances of the organs were fatty and cirrhotic.

Uterus and ovaries normal.

Remarks.—The specimen clearly exhibits a form of pathological change which deserves a distinct place—not only from the standpoint of pathology, but also clinically. From the first point of view we have the structural changes of interstitial cirrhosis in the stage of enlargement (hypertrophic cirrhosis) plus an extensive fatty degeneration of the cell lobules. In simple hypertrophic cirrhosis the liver is hard, brown, and very dark. In fatty hypertrophic cirrhosis the liver is thickened, so that the organ frequently becomes cube-shaped, the masses of connective tissue enclose a yellow parenchyma. The bile-ducts are also commonly implicated in a catarrhal process which results in a choking of the bile-ducts with cells, and the ducts are also commonly surrounded by compact fibrous tissue, and this, with the fatty degeneration of the hepatic cells, constitutes the special pathology of this form of cirrhosis, and suggests the title *Fatty hypertrophic cirrhosis.*

From the clinical standpoint this disease presents distinctive symptoms which may be described under two aspects.

The first of these aspects presents a latent development of the disease, frequently the gradual onset of nausea, vomiting, jaundice, possibly chills, and very irregular fever, in the thermometrical record resembling intermittent fever.

The mental state is that of hebetude, frequently the cholesteræmic state is soon developed. There may be œdema of the limbs and tendencies to hemorrhages from the stomach or bowels. The subjects are usually very fat. The disease runs its course rapidly, and death is apt to occur in six weeks or less.

In the second clinical aspect we have a more prolonged period of incubation as one of the distinctive peculiarities. This period may last from four to eight months. The jaundice develops gradually, there may be diarrhœa, and the stools are apt to be clay-colored; nausea and vomiting are occasional symptoms, but flatulence, pyrosis, and a sense of weight and fulness over the epigastrium, as an evidence of intestinal indigestion, is a frequent symptom. There may be dimness and yellow vision, with languor and mental hebetude. The patient's general appearance in this form of the disorder is emaciated, frequently markedly so, the skin is very yellow, pulse slow and small. Death finally results from prostration, though a semi-typhoid state with slight wandering delirium may be present. There is always a very irregular temperature. Fatty hypertrophic cirrhosis as discovered post-mortem is usually associated with these two sets of clinical symptoms, which, however, possess many common features. In my notes in these cases alcoholism stands first as an etiological antecedent, and repeated attacks of malaria occupy the second place. Finally, from the standpoint of diagnosis, the jaundice and absence of involvement of other organs usually enable one to exclude amyloid disease, a condition which is also excluded by the absence of suppuration in the body; while the smooth enlargement of the liver and the absence of pain can be considered as excluding cancer. *December* 8, 1887.

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3. *Carcinoma of pylorus.*

Exhibited by Dr. J. C. Wilson.

Specimens removed from man in the medical wards of the Philadelphia Hospital.

T. C., white, born in Ireland, laborer, æt. 72 years, unmarried, admitted June 4, 1887.

Two brothers died of pulmonary consumption; family history otherwise good; no history of cancer. Patient was of dissipated habits, and had led a life of hardships and exposure. Had suffered from vague pains about the chest and abdomen for two years. About eight months before admission suffered, for the first time, from distressing gastric symptoms, such as discomfort and sense of weight in the epigastrium after taking food, a sharp pain occurring immediately after taking, and continuing for three or four hours afterward, became a constant symptom.

Upon admission there was great emaciation and weakness, with profound anæmia, the skin being of a dull yellowish color, there being no jaundice.

Patient complained of epigastric pain and prostration; did not complain of hunger, although the amount of food taken was very small indeed; he craved stimulants and narcotics.

There was no epigastric tenderness, nor could any tumor be detected.

The clinical phenomena, beyond the details above given, were wholly negative.

Death occurred Nov. 13, 1887, from asthenia.

External appearances: Body of a tall old man. Emaciation extreme. No *rigor mortis.* Entire absence of subcutaneous fat.

Thorax: Old pleural adhesions; not extensive; both sides.

Heart extremely pale at cortex; small amount of pericardial fluid (5½ ozs. heart); no lesion of valve; anterior wall of heart covered externally by loose, œdematous fat.

Stomach enormously dilated, filled with fluid, small hard lobular tumor at pylorus. No secondary growths. Pyloric end of stomach was bound down to head of pancreas and lesser omentum, and was covered by liver and adjacent tissues, edge of liver not extending below lower margin of ribs. *December* 8, 1887.

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4. *Cicatricial stenosis of pylorus.*

Exhibited by Dr. J. C. Wilson.

B. K., æt. 43 years, laborer, unmarried, was admitted to the Philadelphia Hospital, Oct. 27, 1887. Family history obscure. Patient's previous history mostly negative; stated that he had had occasional attacks of ague.

Began to suffer from gastric symptoms about six months prior

to admission; had sensations of heat and burning pain after taking food, accompanied with waterbrash and heartburn, sensations of weight in the epigastrium, and paroxysmal attacks of copious vomiting at intervals of three or four days, the vomited matters being sour and foul; appetite wholly lost, bowels costive; these symptoms were followed by rapid, extreme emaciation and asthenia. The physical signs indicated enormous dilatation of the stomach, but no epigastric tumor. The clinical phenomena were in other respects negative. Among other methods of treatment, lavage was practised, with temporary relief of the gastric symptoms. Death occurred Nov. 28, 1887.

Post-mortem records of specimens from B. K., æt. 43 years, male, Ireland, Men's Med., admitted Oct. 27, 1887; died Nov. 28, 1887; autopsy forty-eight hours after death.

Body of a moderate sized man, much emaciated, post-mortem discolorations of abdomen, face, neck, and in spots on limbs. On opening abdominal walls, nothing of importance seemed abnormal except that the stomach seemed much distended and very prominent; no adhesions. Intestines quite free from contents, and about region of liver stained with bile.

On opening thoracic cavity, firm adhesions over whole of right lung binding it to the thoracic walls. Small patch of adhesions over middle lobe of left lung.

Pericardium contained the usual amount of fluid.

Heart: Muscle firm and dense, dark in color; over left ventricle a white patch of fibrous tissue, but no adhesions to pericardium. Aortic and other valves seemed competent, with no marked degenerative changes.

No apparent atheromatous change of aorta or coronary arteries, cavities containing chicken-fat clots.

Lungs crepitant throughout.

Stomach enormously dilated and containing about five pints of a dark, sour-smelling fluid, with coagulated cheesy masses and considerable gas. Organ was estimated to be capable of holding at least one gallon of fluid. Pyloric orifice almost completely occluded, admitting only a small probe. On section, the pyloric orifice showed signs of an old cicatrix. About two inches above pylorus, in the greater curvature, were seen evidences of an old, large cicatrix. Mucous membrane of stomach showed post-mortem changes.

Pancreas apparently somewhat enlarged, but normal in appearance.

Liver of normal size, outer surface covered with an old adhesive lymph. Gall-bladder distended with the normal secretions, ducts per-

vious. On section of organ, dark in color, firm, containing dark blood, otherwise normal in appearance.

Kidneys normal in size, somewhat congested, capsules stripped off, cortex in proportion to medulla slightly diminished; no cysts. Other organs, so far as examined, were normal. *December* 8, 1887.

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5. *Disease of gall-bladder and ducts, with gall-stones.*

Exhibited by Dr. J. Daland.

The patient from whom these specimens were removed was under observation only four days, hence this imperfect report.

J. W., male, æt. 50 years, had always enjoyed good health until three or four years ago when he complained of pain and tenderness in the right hypochondriac region with slight jaundice. These symptoms lasted but a week or ten days and then subsided. Since then he has had several similar attacks. It was suspected that the pain was due to the passage of a gall-stone and the stools were carefully searched, on more than one occasion, but none was found. This was the only history that could be obtained when first seen on November 26th, four days before his death. At that time he was distinctly jaundiced, the stools partially colored by bile, the catheter showed the bladder to be empty and this condition of suppression of urine continued until death. He became more deeply jaundiced, pulse rapid, weak, and irregular; delirium, which before was slight now became more marked. These symptoms continued until his death on November 30, 1887.

Autopsy forty-three hours after death.—Skin, mucous membrane, and all the internal structures were tinged with bile. Emaciation moderate. Subcutaneous fat, also fat in the omentum, mesentery, appendices epiploicæ, and about the heart, greater in amount than would be expected. The *gastro-hepatic* omentum was tightly adherent to the under surface of the liver and especially to the walls of the gall-bladder, evidently due to repeated attacks of old peritonitis.

Upon making moderate traction the gall-bladder was ruptured. It contained a small amount of mucus and black bile and three small spherical light-colored calculi were found, about the size of a cherry-stone. The gall-bladder was very much reduced in size, walls thickened and softened. When opening the duodenum a gall-stone, similar in size and appearance, was found in the common bile-duct just at the

point of entrance into the intestines, where it can now be seen. No other stones could be found in the ducts, liver, or intestines.

Examination of the duodenal contents showed that they were composed of pus, blood, epithelium, and bacteria.

The liver was normal in size, rather soft in consistency and greasy to the touch. In numerous places the bile-ducts were dilated and thickened and showed evidence of catarrhal and peritubular inflammation. The kidney showed evidence of chronic catarrhal nephritis.

The bladder was completely empty.

The remaining organs showed no pathological change.

The brain and spinal cord were not examined.

December 22, 1887.

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6. *Perforative appendicitis; general peritonitis. Appendix occluded by a fecal concretion; abscess in the pelvic cavity, surrounding the appendix.*

Exhibited by Dr. J. H. Musser.

I am indebted to my resident physician, Dr. Weston, for the following notes:

J. A. D., æt. 18 years, admitted November 25, 1887. Single, white, worked in a market and lifted a great deal, especially market baskets, which he raised to his shoulders to carry.

Family history and previous history not known.

On November 18, 1887, he ate a large quantity of pies and bananas. While lifting a market basket of fruit on this day (November 18, 1887) he felt a pain in the umbilical region. This did not bother him much, although he had some constant pain until the next Sunday (November 20, 1887), when the pain became quite severe and continued up to the time of death. On admission, he had a very anxious look, quite pale, lips rather blue. Abdominal muscles very rigid. Abdomen scaphoid. Legs flexed on abdomen; breathing both abdominal and thoracic. Pain not markedly increased by pressure, and if attention was withdrawn very firm pressure could be made without noticeable change in amount of pain, and he even said that some of the pain was relieved by pressure. No one spot seemed more tender than another and there was no special spot of hardness or swelling. Urine passed freely with some pain and burning along the urethra and just above pubes. Urine

very dark red, no albumin. It may be stated that during the week he had had three movements of bowels, one after taking a laxative pill and another after taking a bottle of ginger ale. Tongue was coated with a thick yellowish-white coat, great thirst. Pulse very rapid, weak. Temperature 100$\frac{4}{5}$°. Did not rest much on night of admission. This great thirst caused him to drink a great deal of cold water which soon caused vomiting. He vomited, however, only three times during the night.

The next morning (November 26, 1887) patient was in much pain, but not as much as the night before. Hands cold and blue. Lips blue. Face and hands covered with cold perspiration. Heart very rapid, but no pulse at wrist. Mind clear. Abdomen slightly fuller than night before. Moved about bed, flexed and extended limbs without seeming to increase pain. No urine passed during day, but drawn in evening. Vomited frequently during day, nearly every time any medicine was given. Temperature in evening 103°. Died 9.30 P.M. After death considerable blood poured from mouth and nose.

I may add the following additional facts of importance. No tumor nor area of dulness could be detected, and the area of hepatic dulness was well made out. The rectum did not show the presence of invaginated bowel, and the hernia regions were natural. The diagnosis was peritonitis secondary to perforation of the appendix vermiformis.

Post-mortem forty hours after death.—The body that of a well-developed man, eighteen years of age. Abdomen green from post-mortem change. No marks to be noted about body.

Thorax, heart, and pericardium normal. Fluid in pericardial sac, bloody; probably post-mortem.

Lungs crepitant throughout. Some hypostatic congestion in bases and posterior of both lungs. Right lung firmly bound down by old pleuritic adhesions over whole anterior surface and apex posterior.

Abdomen: On opening showed whole contents bound together by recent lymph. From sacs and dependent portion of abdomen, about a quart of greenish-yellow watery fluid was removed. Omentum was bound to intestines and the different coils of intestines all bound together. All these were carefully separated. The head of the cæcum was found to be in the pelvic cavity, where everything was bound together to pelvic walls and posterior wall of bladder. An abscess containining two ounces of pus was found in the pelvic cavity four inches from the anterior spine of the ilium, inside of and one inch below the margin of the psoas muscle and two inches below the level of Poupart's ligament. The appendix was dilated for two inches but normal. At

this distance a perforation, the size of a split pea, was seen communicating with the abscess. The portion of the appendix (one inch long) beyond the perforation, was of a gray color and quite necrosed, soft and collapsed. It was in the abscess. A few lines from the perforation a fecal concretion occluded entirely the healthy duct. The concretion was shaped like a cartridge and its apex pointed toward the necrosed mass. It was one-fourth inch long and one-eighth wide at the base.

Liver slightly fatty. Anterior and upper surface and also under surface of diaphragm was covered by a sheet of recent lymph and pus, greenish-yellow in color.

Kidneys pale, otherwise normal. *December* 22, 1887.

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7. *Chronic ozœna, ulceration of larynx and trachea; cachexia; hœmatemesis and melœna; ulceration of Peyer's patches.*

Exhibited by Dr. W. Osler.

T. S., engineer, æt. 46 years, admitted to University Hospital from Osceola Mills, Pa., on Nov. 30th, in a condition of great debility. Note made as follows: No fever, marked anæmia; is conscious, answers questions; respirations noisy, 16 per minute; pulse, 90–110. On the hands, feet, and buttocks are eruptions presenting the following characters: raised from 2 to 4 mm. above the surface, firm, present a central cap of suppuration, zone of intense hyperæmia; several of these are intensely hemorrhagic, the majority are solid; one on the index finger contained from 10 to 20 mm. of bloody serum; altogether there were 12 or 15 on each hand, and a dozen or more on each foot; some of the medium sized spots are distinctly umbilicated, capped with a small, dry crust. On face and trunk are several smaller spots, resembling closely the drying pustules of varicella. The throat was examined by Dr. Reeves.

Epiglottis looked normal, the arytenoid and ventricular bands on both sides were swollen; below the larynx, on the right side, a dark mass could be seen, extending toward anterior commissure; the extreme weakness of the patient rendered the examination very difficult. From him his physician, Dr. Finley Bell, obtained the following history:

Nothing of note in his family antecedents; never had syphilis; alcohol in moderation. Last spring (of 1887) came under Dr. Bell's care with what was diagnosed as ulcerative ozœna and ulceration of the larynx; is believed to have had ozœna four years ago, on and off; no

signs of lung trouble, and though weak and anæmic, he kept at work. During the summer and autumn he had several attacks of alarming hemorrhage, from, as I suppose, the ulcers (Dr. Finley Bell); he worked up to November 26, 1887, though he needed a stick to help him walk the forty or fifty rods walk between his house and the work. On the 20th had a slight hemorrhage, followed by a severe attack of dyspnœa, which was relieved by the expulsion of two large "scabs"; at this time had a weak, thready pulse, 115; temperature 102° F., soon fell to normal.

The examination of lungs and abdominal viscera was negative, though the area of spleen dulness was increased. There was a soft, systolic murmur at apex. On the 4th he had an attack of hæmatemesis, and passed blood in the stools. There was no fever, no diarrhœa; great prostration, mental hebetude, but no delirium. He sank and died on the 7th.

Cover-glass preparations and cultures were made from the blood and from the eruption, with negative results. As it was suspected that the case might be one of chronic glanders, rabbits were inoculated with the blood and the pus from the spots, but they remained healthy. Briefly stated, the results of the autopsy were as follows: Small ulcer on side of tongue. The left tonsil was covered with a grayish patch. On posterior surface of velum two or three small losses of substance, fresh-looking. No ulceration of, or scars on, pharynx. Some destruction of turbinated bones, but how much was impossible to determine. Epiglottis normal. Right cord swollen, and at posterior attachment covered with gray exudate; erosion of arytenoid cartilage below attachment of this cord. Small inter-arytenoid ulcer, with slough adherent. For an inch and a half below ran a most extensive destructive ulceration, involving the cricoid and the cartilages of the first three tracheal rings. No old cicatrices; no stenosis. Lungs show scattered patches of catarrhal (inhalation) pneumonia. Heart normal; muscle turbid. Spleen was firm, enlarged, and presented a remarkable appearance from the great development of the elements which, in round or irregular patches of gray-white tissue, formed a great part of section. The intestines showed ulceration of the six or eight lower Peyer's patches, the ulcers clean-cut, not extending beyond the patches; the bases made up of the muscle fibres. One presented swelling and congestion of the edges, and a hemorrhagic spot; possibly from this the bleeding came. No special hyperæmia. No swelling of the mesenteric glands. There was an irregular, raised patch of infiltration and erosion at the cardiac orifice of the stomach, involving chiefly the œsophagus.

Cultures made from the spleen tissue did not grow, and no character-

istic organisms could be demonstrated in sections of spleen or of the intestinal ulcers.

The precise nature of this case is by no means clear. The lesions certainly suggest typhoid fever, in which necrosis of the laryngeal cartilages occurs, and according to Cornil and Babes, skin eruptions of an ecthymatous appearance may be present. The absence of bacilli in spleen and intestinal ulcers may be explained on the supposition that we were dealing with the sequelæ of the process. The history, however, as given by Dr. Bell, by no means accords with this view, and it is possible that the entire condition resulted from the chronic ulceration of nose and larynx. In cachectic and debilitated states ulcers in various parts of the gastro-intestinal canal may occur, but I have never, in the adult, seen them confined, as in this case, to the Peyer's patches. The question of syphilis was considered at the autopsy, but the general features of the lesions and the absence of specific history and scars were thought sufficient to exclude it. *December* 22, 1887.

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8. *A case of primary carcinoma of the gall-bladder.*

Exhibited by Dr. G. Dock.

This specimen is from the hospital practice of Dr. Osler, to whom I am indebted for the opportunity of exhibiting it.

It is from a male, æt. 80 years, who was admitted to the Philadelphia Hospital, Dec. 24th. He complained of persistent vomiting and weakness. No satisfactory anamnesis could be obtained. He did not have much pain. Vomiting was uncontrollable, the vomitus being bile-colored. The abdomen was distended and gave fluctuation.

He died of exhaustion on Dec. 29, 1887. At the autopsy the only lesions worthy of note were found in the abdomen. There was no icterus. The great omentum was found rolled up, forming a dense cord. The whole peritoneum was congested and studded with tubercular-looking material. The intestines were matted together so that the whole appearance was that of tubercular peritonitis. There was slight effusion. The mesenteric glands were not enlarged. The liver was fatty. The gall-bladder was contracted, forming a sac 5.5 cm. long and about 1.5 cm. in external diameter. The surface smooth; about the fundus, white and glistening; toward the cystic duct, rougher and discolored. The walls were from 2 to 7 mm. in thickness, dense and pale. The

mucous membrane thickened and the rugæ hypertrophied. The cavity was filled with stones; cystic duct free. About the attachment of the gall-bladder, the surface of the liver was puckered and whitish. Section through this showed a mass as large as a cherry, firm, grayish, not sharply circumscribed. Close to it is a nodule smaller than a pea, firm, yellowish, sharply defined. Chronic nephritis excepted, no other lesions were noted in abdomen or thorax.

Microscopic examination of the walls of the gall-bladder show its mucous membrane hypertrophied. The whole thickness of the wall is the seat of numerous alveoli lined by, or filled with, cylinder or polymorphous cells. In the submucosa these have a structure similar to that of the biliary canals. The nodule in the liver proves to be a scirrhus, the cells in the small alveoli resembling those in the gall-bladder. The thickened omentum is infiltrated with cell nests or fibrous bands containing alveoli filled with characteristic cells.

January 12, 1888.

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9. *Gastric cancer; secondary deposits in the lungs, liver, mesentery and spleen; hepatic abscess opening into the stomach.*

Exhibited by Dr. W. A. Edwards.

F. C., æt. 38 years, Irish by birth, was admitted to my wards in St. Joseph's Hospital, Dec. 29, 1887, and died Jan. 20, 1888.

The patient was unable to give much information either in relation to his family or personal history; beyond the fact that the man was a heavy drinker, we obtained but little information. He had suffered from several attacks of diarrhœa and vomiting within the last year; had an attack of hæmatemesis the day before admission; the blood was bright-red and without odor; a slight hemorrhage occurred on Jan. 5th, and the day before death he vomited a small quantity of blood and passed a large amount by the bowel.

Examination on admission showed marked emaciation, some dulness over the lower epigastric region; no nodules could be felt, but rather a sense of general resistance; jaundice was never present, nor were albumin or tube-casts detected in the urine.

The temperature was normal, neither above nor below the standard; never had a chill or rigor.

Post-mortem was held half hour after death, by Dr. Joseph Otto, the resident. Brain and cord not examined.

Heart small and fatty; all the valves competent; slight atheroma in the mitral curtains, and in the aorta just outside the valves.

Pericardium contained several ounces of serum.

Lungs: Numerous secondary deposits. Right pleural sac obliterated by adhesions.

Abdomen: The stomach large; intestines and left kidney were matted together in an almost unrecognizable mass.

The stomach is much decreased in size, infiltrated, with many nodular deposits, and stenosis of its orifices. The transverse colon is bound directly across the anterior aspect of this large mass, and in one portion is slightly gangrenous. Beneath the diaphragm, and over the dome of the left lobe, is an enormous abscess which has perforated downward through the liver-substance and has opened into the stomach, thus accounting for the hemorrhages; this abscess-cavity contained eight ounces of clotted blood.

The liver shows many secondary deposits.

Kidneys are fatty; ureters, bladder, and prostate glands normal.

January 12, 1888.

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10. *Primary encephaloid cancer of the liver.*

Exhibited by Dr. D. D. Stewart.

G. W. G., æt. 68 years, white, a farmer, was brought to the outpatient medical department of the Jefferson Hospital, on November 21, 1887, for an opinion, by his physician, Dr. Samuel Lovett, under whose care he had been for several months.

His symptoms, as noted in the case-book of the department, were, briefly: Gradually-increasing languor, muscular weakness and emaciation for four months, associated with a painless, rather rapidly growing tumor in the epigastrium, which was first noticed two months after he began to fail in health. There were no gastric symptoms. The digestion was fairly good, the appetite poor, and the bowels moderately regular, though the stools had been persistently pipe-clay colored for one month. The skin had been slightly icteroid for the same time. A slight cachexia had been present for two months. He was anæmic. Physical examination revealed a hard mass, tender only on heavy pressure, which was thought to be the left lobe of the liver, occupying the

epigastric region and a small portion of the left hypochondrium. No nodulation could be made out, probably because of the thickened and œdematous condition of the abdominal walls over the mass.

The liver was enlarged, reaching in the nipple-line superiorly to the third rib, and inferiorly to three inches below the margin of the ribs. The enlargement was irregular, and greater in the direction of the left lobe, which extended across the median line into the left hypochondrium, and downward nearly to the umbilicus.

No enlarged cervical, axillary, or inguinal glands could be found.

His temperature, taken with an accurate thermometer, was 99°, and Dr. Lovett informs me he subsequently found the evening temperature to be constantly above the normal—between 100° and 102°—until a few days before his death, when it became subnormal and so remained.

There was no history of an hepatitis, of a blow, or of the passage of gall-stones, and careful questioning elicited no family history of cancer.

He had not had syphilis or malaria, and there had been no rigors or sweats.

There was a well-marked arcus senilis present, but the heart and vessels were normal.

His urine was free from albumin and sugar; no test for peptone was made. Urates were present in excess, and Gmelin's test showed the presence of bile-pigment.

Nothing more was heard of the case until two weeks ago, when the specimen I show to-night was sent me by Dr. Lovett, with a note giving a few further points in the case and a brief account of the result of the autopsy. The patient died from exhaustion. He had felt no pain at any time until two days prior to his death, when it was complained of in the right hypochondrium. The liver was found to be much enlarged, weighing nine and a half pounds. Large and small nodules were disseminated throughout it, there being very little healthy tissue. There were several large and numerous small masses in the left lobe. The gall-bladder was moderately filled with bile. There were enlarged glands in the portal fissure. None were observed elsewhere, either internally or externally. Nodules in the organs were looked for, but none were found. The other viscera were normal.

The points of special interest in this case are the total absence of pain until two days before death, the presence of but slight tenderness over the liver even on heavy pressure, and the presence of pyrexia for some weeks, perhaps longer, preceding the fatal issue.

January 26, 1888.

11. *Cancer of the mesentery; shortening and matting of the intestinal coil, with simulation of abdominal tumor.*

Exhibited by Dr. W. Osler.

S. A., æt. 28 years, admitted December 22, 1887, with dropsy of abdomen. After tapping, a firm mass could be felt occupying the umbilical and hypogastric regions and extending toward the right Poupart's ligament, immediately above which a solid nodular body could be felt. The case was regarded as one of probable ovarian tumor with ascites. The fluid rapidly reaccumulated and she died early in January, two days after a second tapping. The section showed a sero-fibrinous exudation, the peritoneum slate-gray in color but without any nodules. The intestines formed a compact bunch closely united to the spine, covered with lymph, the coils agglutinated; the whole mass was not larger than a fœtal head, and presented a very peculiar appearance in the distended abdominal cavity. The mesentery was greatly thickened and puckered, and had shortened the intestine in a remarkable manner. The entire bowel was opened *in situ*. The muscular walls were greatly thickened, especially those of the ileum and cæcum; the mucous membrane smooth, and in the ileum thrown into numerous folds on account of the great shortening. There was much thickening of tissues about the cæcum (this was the mass felt above Poupart's ligament), and just within the valve there were two small but deep losses of substance in the ileum which communicated with the end of the cæcum. The appendix could not be found, and possibly these ulcers were connected with past disease of this part. The stomach was normal. The uterus and ovaries and rectum presented no disease. The thickened mesentery on section showed many grayish-white irregular areas which on microscopical examination proved to be of a carcinomatous nature. There was a small angioma of the liver, the portal canals were unusually fibroid, and the round ligament thickened. The thoracic organs were normal. Clinically the case is of great interest, as the coil of closely-adherent, thickened intestine simulated closely a large abdominal growth. The mesentery is not a region likely to be affected with primary carcinoma. Unfortunately the breasts were not examined, and it is quite possible, even at the advanced age of eighty-two, that the original growth was a quiescent mammary cancer. *February* 9, 1888.

12. *Gall-stone at the orifice of the common duct; hepatic intermittent fever.*

Exhibited by Dr. W. Osler.

Woman, æt. 45 years, a drinker, had been in the Philadelphia Hospital on several occasions during the past three or four years with irregular jaundice. She was admitted in January, 1887, and for some months had chills and fever with a variable amount of jaundice. The liver was somewhat enlarged, and she was thought at one time to have abscess. She improved and went out, to return in October with jaundice and pain in the region of the liver. In the course of three months she had four or five attacks of severe pain in abdomen with vomiting, usually a chill, with rise of temperature to 103° to 104°, and a sweat. After the attack the jaundice would deepen. The attacks were so characteristic of Charcot's hepatic intermittent fever, a symptom almost always associated with gall-stones, and considering the protracted course of the disease, I felt justified in urging an exploratory laparotomy, which was performed on the 30th. Perihepatitis and syphilitic gummata had so matted together the parts in the hilus of the organ that it was almost impossible to determine accurately their relations. The gall-bladder was not distended and did not contain any stones. Unfortunately the patient died on the second day after the operation; the condition found I here show. Perihepatitis, old and recent; four or five old gummata, one of which, just above the gall-bladder, had produced considerable deformity of the anterior margin of the organ. In the duodenum a prominence the size of a marble was seen below the mucous membrane, at the papilla biliaria, which represented a gall-stone lodged in the pars intestinalis of the duct. It could be pushed back into the common duct which was moderately dilated. The stone showed facets and was of medium hardness. There was a large-headed gastric ulcer. No lesions of other organs. *February* 9, 1888.

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13. *Hydatid tumor of the liver.*

Exhibited by Dr. G. E. de Schweinitz.

Thomas H., æt. 21 years, white, born in England, and a machinist by trade, was admitted to the Presbyterian Hospital, December 9, 1887.

He came to this country three months prior to the date of his admission. Concerning his previous history and that of his family nothing worthy of note is known. The patient complained of pain over the lower part of the right chest, in front, on deep inspiration. There was marked bulging in that region and in the mid-axillary line. Dulness on percussion extended from the third interspace to three fingers' breadth below the margin of the ribs. Friction-sounds were heard both on inspiration and expiration. There was no movable dulness, and no succussion-splash. Belly tympanitic. Temperature 102.5°. The dulness behind was absolute. An aspirating needle was introduced opposite the seventh interspace behind, at the angle of the scapula, but no fluid discovered. At the expiration of two weeks the patient was able to be out of bed, but was exceedingly listless. On January 5th of this year he became deeply jaundiced, as was shown by the skin, conjunctivæ, and urine. The urine contained bile acids, but no pigment. The bowels were costive, the stools clay-colored, and very fetid. The pulse was rapid, weak, the respiration hurried and shallow. On January 23d a hypodermic-syringeful of opaque fluid was withdrawn, and later nine ounces of pus. This flowed freely for a time, but then ceased and could not be started again, although the trocar was thrust in several directions. Death from exhaustion took place, and the following lesions were found at the *post-mortem* examination: Diaphragm attached to the upper border of the third rib. The middle and lower lobes of the right lung compressed and carnified, upper lobe normal. About one pint of serum in the thoracic cavity. Liver extended from the third rib to four fingers' breadth below the margin of the ribs, and measured 32 cm. from above downward. The right lobe was devoid of liver-substance, except at its margin, and was occupied by a huge cyst, which contained 107 ounces of pus, and three daughter-cysts. The larger cyst was composed of a thick yellowish-white film capsule exteriorly, and within of a semi-transparent membrane, which was deeply bile-stained, besides the contents noted above—in other words, the structures which enter into the formation of a *typical hydatid tumor*. The gall-bladder was full. The common bile-duct occluded. There was considerable perihepatitis, and the capsule was much thickened and adherent to the diaphragm. The spleen was enlarged. The heart and kidneys were normal in structure. The heart was pushed upward, and to the left. The stomach was distended with gas, and much dilated, and pushed to the left. Its long axis was vertical, and the upper border on a level with the nipple. Examination of the fluid revealed pus corpuscles, compound granule

cells, large cells filled with oil-globules, yellowish pigment, cholesterine crystals, and an occasional echinococcus hooklet (three such were found).

Dr. Joseph Leidy kindly examined one of the daughter-cysts, and reports as follows:

The examination of a daughter-cyst, about the size of a large cranberry, discovered granular débris, oil-globules, granular remains of small cysts about the size of sand-grains, but no scolices, hooks, or other remains of scolices. Fragments of the parent cyst had the usual structure, but no remains of attached scolices. This finding, in many respects, corresponds to the so-called *acephalocyst.*

February 9, 1888.

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14. *Chronic hypertrophic cirrhosis of the stomach, with gastric ulcer, colloid change of the mucous membrane.*

Exhibited by Dr. H. F. Formad.

A sailor, æt. 38 years, a German, was admitted to the Naval Hospital ten months ago with the symptoms of dyspepsia, and occasional vomiting. There had been absolutely no pain at any time, and no vomiting of blood. Three months before death he began to have serious difficulty with swallowing, with œsophageal regurgitation, and though a stricture was sought for none was found. Very curiously, for a few days immediately preceding death he swallowed well. Death resulted from starvation. At the autopsy the abdominal cavity was found to contain 52 ounces of straw-colored fluid, and showed evidences of old peritoneal inflammation, especially at the upper part, where the organs were matted together into one mass. The stomach was quite contracted, having a capacity of 4 to 5 ounces. Its walls were ½ to 1 inch thick, the thickness greatest at the cardia, and gradually decreasing till within 2 inches of the pylorus, where the wall abruptly resumed its normal condition. The first impression given was that it was a case of cancer, but closer examination showed it to be simply great thickening of the wall with polypoid elevations of the mucous membrane and secondary colloid change. The ulcer was situated in the posterior wall, near the cardia, its bottom formed by scar-tissue in the spleen and the peritoneal coat of the transverse colon. Communication with the abdominal cavity had been prevented by firm adhesions. The omentum contained a number of colloid granules as large as peas. The abdominal lymph-

glands appeared to be amyloid. The case was remarkable. First: In the complication of hypertrophy with gastric ulcer, a condition which the reporter had never before seen. Second: In that the hypertrophy should commence at the cardia instead of at the pylorus.

February 9, 1888.

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15. *Stab-wound of the abdomen.*

Exhibited by Dr. H. F. Formad.

In this specimen laparotomy had been performed, and a wound of the intestine found, which was sutured. The man died suddenly sixty hours after the operation. At the autopsy there was only a very slight amount of peritonitis found, and the wound in the intestine had firmly united. The abdominal condition not being sufficient to account for death, the brain was examined, and a very distinct embolus in the floor of the fourth ventricle found. The stomach had not been touched, but on its peritoneal surface were some slight tears, and opposite them, in the mucous membrane, but not corresponding exactly with them, were several distinct, small, linear ulcers, which appeared like ulcers due to some injury. They did not look at all like ordinary ulcers, but still had evidently formed ante-mortem. There was an old hemorrhagic infarct of the kidney. *February* 9, 1888.

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16. *Abscess of liver.*

Exhibited by Dr. R. G. Curtin.

R. B., æt. 48 years, by occupation a laborer. Family history is entirely negative, he having left home at an age too early to admit of his being able to give a family history. He had had measles, scarlet-fever, and smallpox, attended by no noticeable sequelæ. As a young man he had no sickness whatever. Had been a hard drinker, and loose in his habits generally. Had had gonorrhœa twice, and also a chancrous sore, not followed by constitutional involvement.

The patient was admitted to the Philadelphia Hospital November 26, 1887, complaining of having been suffering from nocturnal chills for twelve weeks. The chills were irregular, sometimes occurring on several

nights successively, and again skipping one or more nights. During a paroxysm he had great dyspnœa, and could only rest in a sitting posture.

He had a marked prominence on the right side of the chest, just over the seat of the liver. Over this region (once only) he complained of great pain extending around the entire body, with tenderness on pressure, and greatly increased pain on percussion over region of the liver. He had a heavy chill at 1 o'clock A. M., the day following his admission, after which the temperature rose to 103$\frac{4}{5}$°, followed by very profuse sweating. At 7 A. M. it had fallen to the normal point. Another chill occurred the following day, at 4 A. M., temperature 103°, followed by sweating as before, and at 7 o'clock the temperature again became normal. Large doses of quinine were now given, assuming the disease to be malarial in character, as he gave a history of having spent the summer in a Southern malarial district, and as upon the application of a mustard poultice all pain and meteorism had disappeared. The temperature that evening was, however, 101°, with no chill, and did not fall to normal in the morning. At 6.30 P. M. he began to have a chill, and at 7 P. M. temperature was 105°. He was sponged, and antipyretics given, after which the temperature fell to 101$\frac{3}{5}$°. At 2 A. M. another chill was noticed, after which the temperature reached, at 3.30 A. M., 106$\frac{2}{5}$°, and at 5.30 A. M. (two hours after) had fallen to 97° (a decline of 9$\frac{2}{5}$° in two hours).

Two days later, on December 1st, following a similar chill, it rose to 105$\frac{2}{5}$°. On December 3d, at 4 A. M., it reached 106$\frac{1}{5}$°, and on December 5th, at 9 A. M., 103$\frac{1}{5}$°. The temperature after this fluctuated between normal and 103° for four days, when it again rose, on December 9th, at 8 P. M., to 104$\frac{2}{5}$°. It never again reached 101°. A few hours before death it was found to be normal.

Autopsy (*forty hours after death*, by Dr. Osler).—*Thorax:* Slight hemorrhagic exudate in left plura. A few soft adhesions on the right.

Heart: The chambers contained firm, partially-decolorized clots. Endocardium: Blood-stained, walls relaxed, tricuspid segments a little thickened. Mitral segments present a row of small, firm vegetations. No ulceration. Aortic segments healthy. Muscular tissue soft. The right appendix contained a clot, but no globular thrombi.

Lungs: Right crepitant, except a small area in lower lobe, the lowermost part of which is covered with a recent exudate. On section this area was firm, dark in color, chiefly hemorrhagic, centre paler than

periphery, no distinct suppuration. The bronchi leading to this patch were filled with a purulent mucus. In the *left* lung there are two or three of these infarcts, with a pleuritic exudate on the surface.

The main branch of the left pulmonary artery, as it enters the lung, is plugged with a firm thrombus, which extends chiefly into a branch passing to the lower lobe. The thrombus is closely adherent to the wall, and fills the main branch for a distance of two and a half inches. It also passes along the two branches going to the lower portion of the upper lobe. It is grayish-red in color, softened, and in places has a puriform appearance.

On the right a large thrombus occupies the bifurcation of the branches of the pulmonary artery going to the lower lobe. It is firmer than that on the left, and not purulent. Neighboring lung-tissue engorged.

Spleen a little large, brownish-red in color. Pulp a little softened, no infarcts.

Stomach much pigmented at pylorus.

Bile ducts pervious.

The structures in the gastro-hepatic omentum are normal, except that the hepatic artery is unusually large. Right kidney and supra-renal body closely adherent to liver. The diaphragm on the right side is adherent by recent lymph.

Right lobe of liver very much enlarged, right border rounded, capsule opaque. At least one-half of the right lobe of the liver is fluctuating, has a swollen, rounded aspect, projecting upward and to the right. It is very intimately adherent to the right kidney, which is flattened.

Inferior cava laid open: About the orifice of the hepatic veins, coming from the right lobe, there are vegetations, adherent thrombi, and for a distance of one-quarter inch the endothelium is eroded.

Pressure on the liver squeezes a dirty pus, with clots, from the hepatic veins. On further pressure creamy pus exudes.

Transverse section across the liver reveals an enormous abscess occupying fully one-third of the right lobe. Toward the right border the wall is thin, chiefly made up of the thickened capsule, and there is here a distinct lining membrane.

Large intestines: Rectum normal. In the very commencement of the colon and cæcum are cicatrices of four ulcers, and one in the cæcum. They are entirely healed. No ulceration in appendix.

Brain and middle ear not examined. *February* 9, 1888.

17. *Intestinal casts.*

Exhibited by Dr. A. W. Edwards.

The specimen I present this evening has already been reported in *The Medical News*, August 7, 1886, and the following are a few quotations from that paper:

In the literature of the affection we find that the disease has been variously styled by different writers as—tubular looseness or diarrhœa tubularis; chronic pseudo-membranous gastro-enteritis; follicular, duodenal, and clonic dyspepsia; fibrinous diarrhœa; chronic pellicular inflammation of the intestinal mucous membrane; chronic croup of the intestines; mucous disease of the colon; chronic muco-colitis; mucous disease and membranous enteritis; painful affection of the intestinal canal; pseudo-membranous enteritis; intestinal desquamative catarrh; a membrane-like affection of the bowels; intestinal cast.

The following history was elicited:

Susan H., æt. 30 years, light mulatto woman, of good physique, weight 150 pounds, married nine years ago, had one child, born May, 1884, made a good recovery. No miscarriages, or history of uterine complaint. Has always enjoyed good health, is neither nervous nor hysterical, and gives no history of a hereditary taint of any kind. November, 1884, she suffered from a severe attack of dyspepsia, after which first passed an intestinal cast which was quite *fourteen* inches in length, and dark in color,[1] it was thought to be a worm, and patient was treated accordingly; since November has frequently passed casts, suffers no pain during or preceding passage, says that in last six months has passed a "bucketful" of them. No colic, tumefaction, or diarrhœa—in fact, bowels must be kept regular by the use of medicines. Blood never occurs in the discharges. Feels a "sensation of something moving" before cast is expelled; the passage of the casts has been almost constant for the last six months, not occurring in paroxysms. Patient says that she feels well, and were it not for the anxiety that her condition causes her, would consider herself in perfect health.

The disease is generally seen in hysterical women, or hypochondriacal men, who have suffered from dyspepsia and its concomitants. A few cases have been reported as occurring among children.

[1] Specimens were sent for microscopic examination by Dr. Walter F. Atlee, through whose kindness I was enabled to see the patient several times.

Whitehead remarks that out of 100 cases *only four* occurred in males: on the whole, females seem to offer a predisposing diathesis; at all events, cases are more frequent among women.

That physicians and patients may occasionally be mistaken in this disease, witness the following cases:

Quekett (Rich. Quain, *ibid.*) describes the case of a woman who, at intervals of a fortnight or three weeks, had paroxysms of abdominal pain, which were relieved on passing a quantity of membranous matter and tubes, the mass of which sometimes exceeded an orange in size. This mass represented the undigested portion of mutton chops, which had constituted the meat diet of the patient. He states that he has observed *nine* cases of this character.

Corrigan found the substance passed to consist of yellow elastic tissue, resembling the ligamentum nuchæ of sheep.

I cannot better describe the macroscopic and microscopic appearance of these casts than by quoting from the admirable article of Goodhart.[1]

Material consists of: 1st, an elongated network of roundish cords; 2d, white, opaque, solid masses of some inches in length, with small secondary folds on their surfaces, all of which are arranged parallel to the long axis of the mass; 3d, small flocculent pieces of semi-translucent membrane, which, even by naked eye, may be seen to have a corrugated or reticulated arrangement.

The more solid fibres, at their free ends, and on the unattached edges of the secondary folds, oftentimes merge gradually into a perfectly clear and colorless jelly which is hardly visible when placed in water. The cone-like networks have the most consistence, but all three varieties are so delicate as to allow of but very little manipulation out of water.

Microscope: Under two-inch objective, surface is seen to be composed of opaque and translucent parts, the former apparently as round ridges, marking off the latter into regularly-arranged hexagonal or polygonal crypts. Under a higher power these crypts are still visible, though much less well defined. These appearances are best seen in the small flakes of membrane, less distinctly in the larger masses, but not in the networks. They appear to be due to the formation of mucus and epithelial matter, either upon the surface of or in contact with some follicular mucous membrane.

The mouths of the follicles on the surface of the cast, when compared

[1] Casts from the Intestines. By James F. Goodhart. Trans. Path. Soc. London, 1872, xxiii. p. 98, with three illustrations.

with the mucous membrane from a healthy piece of large intestine, present the following differences. They are much larger, in the proportion of two or three to one, while the intervening tissue forming their wall is much less. There is also much less structural arrangement in the cells forming the walls in the membrane passed, and while in the healthy gut distinct basement-membrane exists, no such membrane can be seen in the casts.

Under the least pressure all the specimens break up immediately into isolated collections of cells, these being of various shapes and sizes; but the greater number are elongated, and blunt at one end, having the appearance of disintegrating columnar epithelium. On making a section vertical to the surface, and being careful to make no pressure with the cover-glass upon the specimen, indistinct pieces of tubes or their moulds may here and there be seen, but only once or twice were these very distinct. All stain deeply with carmine, in inverse proportion to their opacity, gelatinous parts staining most deeply. Chemically, all are dissolved by alkaline solutions, by strong hydrochloric acid, and heat.

But few post-mortems of the disease have been recorded. Simpson mentions a case, seen by Abercrombie, in which the mucous membrane of the colon was covered by an immense number of small spots of a clear white color, which were vesicles that, when punctured, discharged a small quantity of clear fluid. This patient had suffered from phthisis, and had passed a large quantity of "membranous casts or tubes."

DaCosta is of the opinion that the affection is not originally an inflammation, but considers the inflammatory element as the result rather than as the cause, and would attribute the true etiology to the nerves presiding over nutrition. In Wright's case the mucous membrane of the colon and lower portion of the small intestine was studded with a thickly-settled papular eruption.

The colon seems to be the seat which the disease elects; this, however, is not the invariable rule. The middle period of life also seems to be the age at which the passage of intestinal casts most frequently occurs. Again, however, this is not without exception; as has been stated, a few cases have been met with in children.

Intestinal casts may be mistaken for dysentery and fatty discharges; again they have been mistaken for worms. In diphtheria the intestines may be attacked by diphtheritic membrane, and the casts passed per rectum. An extremely interesting case, showing how diphtheria may present casts of a portion of the alimentary tract, is recorded in a late number of the *Amer. Journ. of Med. Sciences.*

In pyæmia, scarlet fever, puerperal fever, and tubercular disease, a membrane is sometimes deposited and cast off. These are cases of "secondary croup of the intestinal membrane." *February* 9, 1888.

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18. *Specimen of epithelioma of pylorus.*

Exhibited by Dr. F. P. Henry.

Although this specimen has not been examined under the microscope, I feel reasonably certain that it is one of cylindrical-celled epithelioma. I do not, however, wish to forego what I believe will be the corroborative evidence of the Committee on Morbid Growths. My reasons for believing this tumor to be a cylindrical-celled epithelioma are derived rather from the clinical history of the case than from the appearance of the growth, for an ulcerating carcinoma might present the same ragged, uneven surface. If this appearance, however, had been due to ulceration of a growth in this situation, there would have been a history of pyloric stenosis, with consequent gastric dilatation and vomiting, followed, in the ulcerative stage, by pyloric incontinence, with passage of the fermenting and putrid contents of the stomach into the intestine, and diarrhœa. These are the usual symptoms of such a pathological process, the existence of which is disproved by the facts that there never was any vomiting in this case from first to last, and that the condition of the bowels was one of chronic constipation.

The patient was a man æt. 30 years, an inmate of the Philadelphia Hospital, who, when he came under my care, on February 18th, was in a state of extreme prostration and emaciation. The abdomen was markedly scaphoid, and so tense that palpation gave no positive results, except tenderness on pressure in the epigastrium, and a sense of resistance in the same region. The combination of extreme tension of the abdominal integument with the greatest emaciation, and without tympany, possesses, I think, some diagnostic significance in cases of abdominal cancer. As there was no vomiting, the contents of the stomach were not examined chemically, for the man was altogether too weak to justify the use of lavage for the mere purpose of obtaining some of the gastric secretion. The state of this man's blood was such as to be of decided diagnostic value. The only disease with which cancer of the stomach, without vomiting, is apt to be confounded, is pernicious anæ-

mia, and this I was enabled to exclude at once by my examination of the blood. On February 7th I found the number of red corpuscles per cubic millimetre to be 2,400,000, which is a number very greatly in excess of that which would be found in the last stage of pernicious anæmia. The white corpuscles numbered 15,000 per cubic millimetre, which makes their proportion to the red as 1 to 160. They are both absolutely and relatively increased. The amount of blood necessary for Gowers's hæmoglobin test (20 cubic millimetres) could not be obtained from the finger even after a deep incision. Numerous "granule masses" were present, and as these are made up of blood-plaques, which are the chief factors in thrombosis, the fact is one of great interest in its relation to this case, for venous thrombi had formed in both lower extremities.

The tumor, as you see, forms an ulcerative thickening of the pyloric end of the stomach, and extends inward for a distance of two or three inches, and although it involves the lumen of the pylorus it has not produced any stricture of that orifice.

The morbid growth has not given rise to any secondary deposits, except in a few lymph-glands in the gastro-hepatic omentum. The extreme wasting of this patient's tissues is indicated by the fact that the liver, stomach, and spleen together weighed only 2 pounds 15 ounces.

Report of the Committee on Morbid Growths: Your Committee reports that the specimen exhibited by Dr. F. P. Henry presented the appearances of an ulcerated glandular-celled carcinoma.

February 23, 1888.

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19. *Specimen of bothriocephalus latus.*

Exhibited by Dr. F. A. Packard.

This specimen was passed by a patient in the men's medical ward of the Pennsylvania Hospital last summer. He was a native of Finland, and could speak no English. He was brought to the hospital from a British steamship, the captain of which stated that he had been suffering from diarrhœa for the several preceding weeks. He was found to have a profuse diarrhœa, with greenish-brown, semi-liquid movements. He was given (a few days after his admission) f ℥ss of castor oil. In a few hours after its administration the specimens shown were found in the movement. On examination these were found to be evidently parts of

two worms, as manifested by the appearance of the size of the links. The heads could not be obtained, although the segments dwindled down to minute specks. There are seen to be rosette-shaped uteri and the large oval eggs. No other segments were passed. The course of the diarrhœa remained unchanged after the voiding of the worms.

March 8, 1888.

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20. *Follicular enteritis.*

Exhibited by Dr. A. V. Meigs.

A foundling brought to the Sheltering Arms, December 26, 1886, by a policeman, in bad condition, with eczema of the head and sore buttocks. She was taken to be about three months old. While in the house she had no vomiting, nor diarrhœa, but the movements were somewhat irregular, sometimes being green and again natural. The infant, however, never picked up much. There was whooping-cough in the institution, and for about ten days before death she had some cough, but it was not very severe. She had very severe pain in the abdomen, apparently, for a day before death, which occurred February 17, 1887. Post-mortem made about twenty-four hours after death. Heart not opened, but appeared natural; was firmly contracted. Lungs rather pale flesh-colored, and everywhere crepitant, except small areas upon posterior surfaces, where there were several spots a quarter to half an inch across, which stood out from the general surface and were dark-blue in color, hard, and non-crepitant. Liver very dark and filled with blood. Spleen appeared natural. Kidneys looked natural, but several minute concretions were found in one of them upon section. Stomach exceedingly small, its capacity could not have been more than an ounce; but internally it appeared natural, containing only some very viscid slime. The mesenteric glands were much enlarged, some being of greater diameter than a large pea. In both small and large intestines there were areas where the walls were exceedingly thin, the mucous coat seeming to have entirely disappeared, and such areas were almost transparent, again the mucous coat could be seen still *in situ*. The first Peyer's patch below the stomach was very greatly thickened and dark-red in color, showing upon the outside of the gut as well as within, the surrounding portion of the intestine was very thin and transparent, the mucous coat seeming absent. Lower down many of the Peyer's patches showed in

a characteristic manner the shaven-beard appearance, and again others were ulcerated and had raised and everted edges. In the large intestine areas which were exceedingly thin and transparent alternated with spots much thicker upon which the mucous coat could still be seen. There were many minute dark spots which looked like healed ulcerations of solitary glands. *March* 8, 1888.

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21. *Enteritis and acute hydrocephalus. Left olivary body smaller than right.*

Exhibited by Dr. A. V. Meigs.

An infant taken to be about ten weeks old was brought to the Sheltering Arms, February 8, 1888. He was deserted by his mother about one week before, being left at a boarding-house. When brought to the institution he was in bad condition, having been fed upon milk with corn-starch. After admission he was given milk-and-water (1:3). The bowels were loose and he vomited. While in the house his condition varied, and he was fed upon barley-water and milk for a while, and at one time it was thought he improved, though the bowel movements continued to be green. He had been operated upon for phimosis before admission, and previous to death there was a great deal of inflammation and swelling of the penis. During the last three days of life he would occasionally, without warning, cry out in a sudden shrill way (the cry of hydrocephalus). He died February 29, 1888, aged about three months. The post-mortem showed the lungs to be generally pale, but there was marked hypostatic congestion of the posterior dependent portions of both of them from apex to base, and here and there upon the surface were scattered dark spots of collapse. Heart natural. Liver dark in color and much congested, the blood running from it very freely upon section; otherwise natural. Bile-duct patulous. Spleen natural in size, appearance, and consistence; on section the Malpighian bodies not very marked. Kidneys natural. Stomach contracted, having a capacity of about two ounces, its inner surface presenting no abnormal appearance. Mesenteric glands very much enlarged. Small intestine everywhere much injected. At the middle portion the inner surface was covered with a thick, slimy material, which adhered closely to the mucous surface, and was either a very tenacious slime or pseudo-membrane. (After being kept in alcohol this shrivelled and came off.) At the lower portion the Peyer's patches were often very thin at their cen-

tres, and had thick and everted edges. The large intestine was thick at some portions and thin at others. In areas there were minute, dark, ecchymotic-looking spots, which must have been the remains of healed ulcerations of the solitary glands. The brain was exceedingly soft, almost diffluent, so that it was impossible to remove it without tearing. The lateral ventricles, which were opened before removing the brain from the skull, were found to be dilated, having a capacity of one ounce each, and were filled with serous fluid. The posterior horns were relatively more dilated than the anterior, the fluid having gravitated backward. The brain-substance was congested, being more pinkish in color than natural. At the base the meninges were much more highly colored and injected than natural, and seemed entangled over the pons and medulla oblongata, so that it was difficult satisfactorily to distinguish their outlines. The olivary body upon the left side was seen to be decidedly smaller than that upon the right, which latter appeared to be natural in size and shape. Spinal cord not examined.

March 8, 1888.

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22. *Tubercular disease of cæcum and colon; chronic phthisis; typhoid state.*

Exhibited by Dr. William Osler.

Max S., æt. 45 years, admitted to the Philadelphia Hospital January 21, 1888. Stated that he had been ill for three weeks. Had been gradually getting weak, and losing flesh and strength, and has had diarrhœa and fever. Was previously healthy, and worked up to onset of present illness. On 22d, when first seen, was in a very weak, debilitated condition, looking like a man who had passed through an acute illness. Pulse 120. Temperature 99° in the morning, 101.2° in the evening. Tongue coated and dry. Abdomen soft, a little swollen, tender in right iliac fossa. Four inches of splenic dulness. Dr. Bradley thought he noticed one or two suspicious spots. From this date until his death, a week later, he was delirious, extremely feeble. Pulse 120. He had two or three stools each day. On the 24th and 25th he had chills, after which the temperature rose to 103°. He had no cough, no expectoration. The heart-sounds were scarcely audible, no murmur. There were a few râles at both apices. The autopsy showed localized tubercular peritonitis over ileum, cæcum, and ascending colon. The ileum presented four or five ulcers, one near the valve very large. The upper part of cæcum and the first three inches of ascending colon

were extensively involved. The walls were much thickened, and this portion of the bowel formed a firm, solid mass. The lumen was greatly narrowed, barely admitting the little finger. Laid open, it presented numerous ulcers, the bases of which were distended as sacculi containing little balls of feces. The ridges separating the ulcers presented elongated, polypus-like extensions of the mucosa. In the neighborhood of the uppermost ulcer tubercles could be seen. No other ulcers in colon. There was old tubercular pericarditis, with great thickening of both layers. Lungs were crepitant except at the apices, both of which presented small cavities, puckered cicatrices, and fresh tubercles.

March 22, 1888.

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23. *Calcareous bodies.*

Exhibited by Dr. J. H. Musser.

These small, hard, irregular bodies (eight) were found behind the cæcum in the connective tissue. They were intimately surrounded by tissue; in fact, it was hard to shell them out of their covering. They vary in size from a pea to an almond. They are evidently calcareous, and the addition of hydrochloric acid causes solution with effervescence. They are probably glands which have undergone calcareous transformation. The illness that caused his death had no bearing on their development, and nothing could be ascertained of his previous medical history to give a clue to their origin. *March* 22, 1888.

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24. *Colloid cancer of the stomach.*

Exhibited by Dr. J. H. Musser.

Neither orifice of the organ is involved. The growth includes the entire circumference of the stomach toward pyloric end. Its right edge is two inches from the pylorus; its left six inches from the œsophagus. It occupies from five to seven inches of the entire length of the stomach. The mucous membrane and the submucosa are involved entirely in the disease; the muscular coat is invaded in a few places; the peritoneum is free from infiltration, though opaque in certain areas, and adhesions attach the mass to the left lobe of the liver. The inner surface appears as a large, irregular ulcer, with thickened edges and massive broad and

flattened projections on the floor, which almost occlude the lumen of the cavity. The surface of the ulcer is covered with mucus and blackened blood, and in various parts small recent hemorrhages have occurred, while on close scrutiny the alveolar structure, peculiar to colloid cancer, is observed. That portion of the stomach that is situated between the œsophagus and the diseased mass is dilated, and its mucous membrane congested.

The tumor was found in the median line of the abdomen, midway between the xiphoid cartilage and the umbilicus. Two-thirds of the mass was on the left side of the median line. The peritoneum, mesenteric, and retro-peritoneal glands, and all the abdominal organs, were free from malignant disease. The abdominal cavity alone was examined.

Microscopical examination revealed the characters of colloid cancer.

The patient from whom the specimen was removed was a female, æt. 52 years, married, the mother of eleven children. She had used malt liquors in moderation, lived on the diet commonly used by the middle class of Germans, worked very hard, and, though never ill, suffered in later years from dyspepsia. During the entire illness, about thirty months, the clinical features common to cancer of the stomach were observed—pain, dyspepsia, vomiting, hemorrhage, constipation, emaciation, and tumor. The tumor was superficial, hard, and always more or less movable. A peculiar feature of the last three months was the presence of sialorrhœa. The flow was excessive and exhausting.

March 22, 1888.

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25. *Macroglossia.*

Exhibited by Dr. H. R. Wharton.

The subject from whom this specimen was removed was a child æt. 5 months, a patient in the Children's Hospital. The hypertrophied condition of the tongue had existed since birth, and a portion of the tongue was always protruded from the mouth. A V-shaped portion of the tongue, including the top, was removed, and the edges were then brought together with sutures, and firm union had occurred at the end of ten days; at this time the organ could be well retained within the cavity of the mouth. The child was perfectly well, but its friends, who resided some distance from the city, failed to remove it from the hospital when notified so to do, and some weeks afterward, during a spell of

intensely hot weather, the child suffered from a heat-stroke which proved fatal. The results of the post-mortem examination and the microscopical appearance of the section of the tongue removed will be described by my colleague, Dr. de Schweinitz.

This interesting and rather rare condition of hypertrophy with prolapse of the tongue, which is generally known as macroglossia, may be due to several causes.

First. To a true hypertrophy of the muscular elements of the tongue, which seems to have been the condition which existed in the case I have reported.

Second. To an hypertrophy of the lymphatic elements of the tongue.

Third. To an angeiomatous condition of the vessels of the tongue, or both the vascular and lymphatic elements may be involved, giving rise to a lymph-angeioma of the organ.

If the disease is marked the patients suffer from difficulty in breathing and in taking food, and the shape of the alveolar processes of the jaw may be changed by the pressure of the hypertrophied organ.

The treatment of the condition may be by amputation of a portion of the tongue, or, in suitable cases, removal of a wedge-shaped portion of the organ and the bringing together of the remaining edges by sutures; as the result of either operation there is generally a marked diminution in the remaining portion of the tongue.

Notes on the post-mortem of Dr. Wharton's case of macroglossia, and microscopic examination of the tongue, by G. E. de Schweinitz: More than a month after the operation this child died from the effects of acute pyrexia or heat-stroke. On the day previous to its death no untoward symptoms were noted, and the night was passed with no more apparent discomfort than would be accounted for by the very warm weather. Between seven and eight on the following morning the child was asleep, but two hours later the respiration became labored, the face at first flushed and later cyanosed, the hands and feet cold, but the body very hot. The thermometer in the axilla registered 110° F. Treatment consisting of cold packs, hypodermics of antipyrine, and during marked respiratory failure atropine was instituted. The temperature by degrees fell to 100° F., but the coma, which had been present from the beginning, did not disappear, and the little patient gradually sank, and died about 2 P. M.

Autopsy, six hours after death: Body of a well-nourished baby. Slight rigor mortis—especially about muscles of the neck.

Thorax: Lungs everywhere crepitant and normal in appearance, except for slight congestion of their bases. Pleuræ smooth.

Heart and pericardium: The usual fluid in the pericardial sac. The heart normal in size; the left ventricle tense and *firmly contracted.* The valves normal. The blood dark colored, fluid, and markedly acid in reaction.

Abdomen: Liver-tissue firm, dark, and congested.

Spleen slightly enlarged, pulp soft and dark-purple.

Stomach and intestines: Stomach nearly empty—a few slight submucous ecchymoses. Through the entire length of the small intestine the solitary and agminate glands were greatly swollen. This was particularly noticeable in Peyer's patches, where the enlarged lymph elements, the injection of the permeating capillary network, and a surrounding zone of congestion, made a beautiful demonstration of these tissues. The rest of the abdominal and the pelvic organs appeared normal.

Brain and membranes: The veins of the convexity were filled with dark-colored blood. The membranes were smooth and devoid of any spots of opacity. There was slight sub-arachnoid effusion. The anterior lobes were slightly flattened and the lateral ventricles somewhat distended with liquid. The puncta vasculosa was unduly prominent and of a dark-red color. Upon the posterior portion of the left optic thalamus, as well as upon the surface of the hippocampus major, were a number of small extravasations varying in size from a millet-seed to that of a split pea; the larger ones being upon the optic thalamus. No other gross lesions were discovered in the brain. On removal of the posterior half of the right eyeball a small dark spot was noted upon the outer side of the disk—presumably a retinal hemorrhage. This was reserved for microscopical examination.

Microscopic examination of the excised portion of the tongue gave the following results, from without inward: 1. The mucous membrane, consisting of its layer of connective tissue, supporting the papillæ and covered with epithelium. 2. Longitudinal and transverse bundles of striped muscular fibre, with delicate films of connective tissue between them. 3. Collections of racemose glands, in cross-section, between the muscle bundles. Congenital and infantile hyperplasia of the tongue, or *macroglossia*, consists, as is well known, of an enlargement due to overgrowth of all the constituent tissues of the organ, or of the connective tissue only, or, it may be, to the development of neoplastic tissue, including the condition known as *lymph-angiectatic macroglossia*, or that disease which depends upon a dilatation of the lymphatics. In the case presented this evening there was an overgrowth of all the constituent tissues of the organ; in other words, a simple hypertrophy.

March 22, 1888.

26. *Cirrhosis of liver.*

Exhibited by Dr. Bailhache.

Wm. D. B., seaman, native of West Indies, admitted to Hospital Feb. 23d, suffering with acute hepatitis followed in a few days by jaundice; was in hospital the month previous with scalded hands, and discharged cured January 28th. Died March 29th, after several days' coma. A clinical history indicating yellow atrophy, and a post-mortem indicating cirrhosis. Body well nourished, fat, *very* yellow—a real golden hue—stools colorless, region of liver only slightly painful on pressure, not enlarged at any time and rapidly atrophying, particularly the right lobe, which could not be found without difficulty, No ascites, no œdema, no great distress complained of. Intellect became cloudy about March 17th, and unable to distinguish small objects. Very offensive breath. Tumor detected on left side, but disappeared a few days before death.

March 22, 1888.

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27. *Specimens from a case of typhoid fever, with limited intestinal lesions.*

Exhibited by Dr. G. Dock.

These specimens are from a case under the care of Dr. J. P. C. Griffith, to whom I am indebted for the opportunity of presenting them:

T. T., æt. 22 years, ironworker, born in Canada. Admitted to Dr. Pepper's wards in the University Hospital, March 29, 1888.

Family history good. With the exception of measles and whooping-cough in childhood, has never had any illness until present. In the beginning of December, 1887, he lost his job and became a tramp, and has never felt well since that time.

Three weeks before admission noticed that he was drowsy, felt weak, and this was soon increased by the occurrence of diarrhœa. About that time there was pain in back and abdomen and loss of appetite. He took to bed March 26th.

Stat. præs., March 29, 1888. Well-developed man, of average size, sensorium cloudy, face flushed, tongue glazed, red, and beefy. Abdomen slightly distended. No œdema.

Lungs: Percussion normal. Fine râles both sides.

Heart: Percussion dulness diminished. Faint systolic murmur at apex, not transmitted.

Liver enlarged.

Spleen cannot be felt. No gurgling in right iliac fossa.

Urine: Specific gravity 1035. Examination negative.

Temperature on admission was 104.2° F.; pulse 104; respiration 28.

Two days after admission four spots appeared on abdomen. There was epistaxis, headache; temperature reached 105° F.

April 2. Three more spots; tenderness and gurgling in right iliac fossa. Very drowsy; subsultus and carphologia.

4*th.* Very delirious during night. Tried to leave bed. Spleen could be felt below costal margin.

6*th.* Eyes half open; face pale; tongue dry, brown; sordes on teeth. Abdomen somewhat distended. Pulse weak and running, 140; temperature 104° F. Involuntary passage of urine.

On the 7th temperature ran up to 106° F., and exitus ensued.

On post-mortem examination there was found intense inter-alveolar emphysema; heart presented no marked changes—no ante-mortem clot. Spleen three times normal size, dark-red, soft.

The intestines were stained with bile in their whole extent. No lesions were found until within two feet of the ileo-cæcal valve. In that part are three small Peyer's patches which are ulcerated, with thick walls. The mucous membrane and submucosa are infiltrated and congested, and scattered through the affected part are many small, round or irregular ulcers with greatly infiltrated walls. In the remaining part of small intestine, the appendix and colon there are no enlarged solitary nodules, and in the affected part are numerous nodules not swollen. Other organs, including brain, show no lesions. The mesenteric glands are moderately swollen.

Microscopical examination of the gut shows intense infiltration, necrosis of epithelium, lymph spaces in muscular and subserous layers engorged with cells. *May* 10, 1888.

28. *Cirrhosis of the liver.*

Exhibited by Dr. D. J. M. Miller.

The notes of this case were unfortunately misplaced; but the history, as far as can be recollected, is briefly as follows:

The patient, a woman, æt. 40 years, was treated at the Episcopal Hospital two years ago for ascites and œdema of the lower extremities. She remained in the house four months, and was discharged much improved. She was admitted again last December by Dr. Fisher. At that time moderate œdema of the legs and ascites existed. There was an aortic, systolic, and diastolic murmur. The urine was non-albuminous. The liver measured 3 inches in the nipple-line. There was a large patch of lupus erythematosus on her face. She had vomited blood several times during her illness, and had voided coffee-ground matter by stool. She was not intemperate Under treatment the ascites and œdema disappeared, and she was fairly well when the ward was transferred to my care on April 1, 1888. A week later she died from facial erysipelas.

At the autopsy this typically cirrhosed liver was found. The organ weighed 2 pounds. It was supposed by several who saw it to be syphilitic, but Dr. Simes, who kindly examined it microscopically for me, found no ground for such a belief. The gall-bladder was greatly distended and filled with a perfectly colorless fluid. The cystic duct was completely occluded. There was a marked narrowing of the aortic orifice. *May* 10, 1888.

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29. *Corrosive sublimate poisoning.*

Exhibited by Dr. H. F. Formad.

The exact time that had elapsed between the ingestion of the poison and death was unknown. But it was known that an hour before death the patient was perfectly well and the poison must have been taken subsequently to that time. There was an intense congestion of the mucous membrane of the stomach confined to the cardiac end, the remainder of the stomach being comparatively unaffected. The heart was hypertrophied. The kidneys large and pig-backed. *May* 10, 1888.

30. *Perforated vermiform appendix removed by operation from a case of peri-cæcal inflammation.*

Exhibited by Dr. D. J. M. Miller.

Jacob L., æt. 19 years, car-driver, American, single, was in good health until Saturday, April 28, 1888. On that day, while jumping over a hurdle, he felt a severe pain in the lower abdomen, which in a few moments passed away. The next day diarrhœa came on and continued until May 8th. During this period he felt quite well, except occasional colicky pains. Suddenly, on Tuesday, May 8th, there occurred severe pain in the right iliac region, shooting down the right thigh and leg, and increased by walking or any movement. Simultaneously with the onset of this pain the diarrhœa ceased. Vomiting occurred several times. On Thursday, May 10th, at 5 P. M., he was admitted to the Episcopal Hospital. I saw him for the first time at noon the next day. He was then lying on his back with the right thigh flexed and complained of constant aching and stabbing pains over the right iliac region, with shooting pains and numbness of the corresponding lower limb. His face was anxious and indicative of great suffering. His temperature the night before was 102° F., and was then about 101° F. The tongue slightly coated; bowels constipated. Examination showed a fulness of the right iliac region. The abdominal walls over the area were motionless. Palpation, which was exquisitely painful, revealed at a point 2 inches above the middle of Poupart's ligament a marked sense of resistance, and a dull area about 2 inches square. This was not so apparent when the leg was strongly flexed. Rectal examination was negative. Eight Swedish leeches were ordered to the painful part. The next day, May 12th, he was much better, great relief having followed the leeching. His temperature was 99°; it had been 102° the evening before. The pain was not constant, and the limb but slightly flexed. The dull area, which was still very painful, percussion causing flexion of the thigh at every stroke of the finger, still persisted. Dr. Nancrede, my colleague in the surgical wards, then saw him and coincided with the diagnosis of a localized peritonitis probably due to appendicitis. At his suggestion, to eliminate the possibility of a stercoral typhlitis, Epsom salts in ʒj doses was ordered to be given every two hours until the bowels were freely opened. There were no motions until 5 P. M. the next day, or until ʒjss of salts

had been administered, half a dozen small watery stools then occurred. His temperature at 5 P. M. on the 12th was 102°; the next morning about 100°; that evening 102⅕°. The local condition remained the same. On Monday, May 14th, at noon, there was some increase of pain, and the area of dulness had slightly enlarged. Dr. Nancrede then saw him again, and it was decided should there be any further increase of the local symptoms, an exploratory incision should be made. These conditions seemed to be fulfilled that evening, and the abdomen was opened by Dr. Nancrede with the consent of the surgical staff at 11 P.M. At the operation the transversalis fascia and peritoneum were found thickened and inflamed; the appendix was invested with a thick layer of lymph, and its extremity so softened that it was torn in its removal. The area about the caput coli was shut off from the abdominal cavity by layers of thickened lymph, and contained an ounce or so of fecal-smelling serum. The appendix was ligated close to the bowel. The patient did well after the operation, with the exception of the second day, when, on account of the presence of some signs of peritonitis, he was purged freely with salines. He is now practically well.

On examining the appendix it was found to be covered with a layer of thickened lymph, easily stripped from the gut. On slitting it up the mucous membrane seemed to be in a catarrhal condition, while at a point about one inch from its distal extremity there were distinct signs of softening and loss of substance; at this place, also, there were three distinct perforations which communicated freely with the mesentery, and which had evidently commenced in the follicles of the mucous membrane. The whole of this portion of the appendix was soft and friable.

Appendicitis in the great majority of cases is due to the presence of some foreign body or to fecal concretions. In this case no such cause could be found, and the cause must be sought in an extension of the intestinal catarrh from which the patient was suffering. The sudden onset of pain on May 8th was evidently the date of the perforation. The necessity of an early operation in these cases seems to be clearly indicated by the specimen exhibited. The operation was performed before suppuration had taken place. There is little doubt that in forty-eight hours the appendix would have become gangrenous, or that with or without that occurrence general peritonitis might have supervened.

May 24, 1888.

31. *Encephaloid carcinoma of stomach and pancreas; metastasis in liver.*

Exhibited by Dr. D. J. M. Miller.

Isabella D., æt. 42 years, white, unmarried, housemaid, native of Ireland, was admitted to Episcopal Hospital, April 23, 1888. Both parents dead; cause unknown; one brother died of phthisis; a brother and sister living and healthy. Has had smallpox, and for the past twelve years has suffered greatly from dyspepsia, which she attributes to excessive tea-drinking. Symptoms of present illness began six months ago, it has been characterized by occasional vomiting and epigastric and hypochondriac pain. Blood has been vomited some three or four times; once, three weeks before admission, it was copious and of a bright-red color. Has also passed dark "coffee-grounds" material per rectum. Vomiting usually occurred after eating, and consisted of undigested food. On admission the patient presented a marked cachectic appearance, and was greatly emaciated; tongue coated, appetite lost, bowels constipated; severe stabbing pain through epigastrium. The latter region was tender to pressure and tympanitic on percussion. The condition of the right lobe of the liver, owing to tympany and tenderness, could not be satisfactorily determined. Over the lower part of the chest in front, in the so-called semilunar space of Traube, there was dulness extending one inch below the margin of the ribs, from this point to within one inch of the umbilicus the note was clear and tympanitic; about the umbilicus there was an obscure dulness with increased resistance, and great tenderness on the slightest touch. The lungs seemed healthy, except feeble breathing over the left base, laterally and behind. There was a suppurating gland in the left axilla. She vomited very little after admission, and then only curdled milk. Pain was intense, and could only be relieved by large doses of morphine. The umbilical tumor gradually increased in size, and by May 5th measured four inches in diameter. The greater part of its area was to the right and above the umbilicus. On palpation it seemed to give an obscure sense of fluctuation; but Dr. Nancrede, my colleague in the surgical wards, withdrew nothing but a little blood on introducing a hypodermic syringe into its centre. During the last two days of her life she vomited continuously. She died on the 13th of May, twenty days after admission, and almost seven months from the onset of active symptoms. She never vomited blood, nor passed any per anum after entering the hospital, nor was there ever any elevation of temperature.

Autopsy, made twenty-four hours after death.—Abdominal wall was found firmly adherent to the mass at the umbilicus, and on its removal part of the tumor contents escaped. It was situated just below the pylorus, and its cavity, which was filled with semi-fluid pulpy matter and clotted blood, and which was separated from the general peritoneal cavity by a firm investing membrane, extended to the left and upward behind the posterior wall of the stomach. On opening that organ there was found on the posterior wall, at the lesser curvature, one and one-half inches from the pylorus, a tumor the size of a walnut. It was of the same consistence as the external mass, was easily broken up by the finger, and communicated freely with the interior of the growth first mentioned. The body of the pancreas was also involved. There was no fluid in the peritoneal cavity. The liver weighed four pounds and two ounces, and its fissure was a little to the left of the median line, so that the enlarged right lobe filled up the left hypochondrium and caused the dulness in this region noted before death. On the anterior border of the right lobe there was a large fluctuating umbilicated growth, the contents of which resembled those of the tumors above mentioned. Similar growths were situated in the upper right border of the same lobe, while almost the whole of the left lobe was broken down and infiltrated by the morbid growth. The left lung was congested at its base; the right was normal. The spleen was small in size, and its interior softened and filled with dark blood. The kidney seemed to have undergone some fatty change. The heart and other organs appeared healthy.

In this case the growth doubtless originated in the stomach, ulcerated its way through its posterior wall and infiltrated the tissues back of that organ. The liver growths were probably secondary. A microscopic examination of the growth by Dr. Simes showed them to be encephaloid carcinoma.

For the notes of this case, as well as for making the autopsy, I am indebted to Dr. A. A. Ghrisky, the resident physician.

May 24, 1888.

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32. *Duodenal ulcer.*

Exhibited by Dr. H. F. Formad.

During life, until within a few hours preceding death, there had been no symptoms. Then the sharp pain and vomiting which set in aroused

the suspicion of poisoning. But at the autopsy a sharp-cut duodenal ulcer was found. This ulcer had perforated, leading to the escape of the contents of the intestine into the abdominal cavity. The time between the perforation and death had been so short that no peritonitis was set up. *June* 28, 1888.

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33. *Gastric ulcer.*

Exhibited by Dr. H. F. Formad.

This gastric ulcer was found in an insane patient æt. 35 years. Death took place during very hot weather from the exhaustion following an attack of mania. During life there were no symptoms pointing to gastric ulcer. Dr. Formad referred to the frequency of gastric ulcer in the insane, and also to a large series of cases in which the ulcers had not caused death, nor had they shown their presence during life.

June 28, 1888.

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34. *Tubercular peritonitis.*

Exhibited by Dr. M. H. Fussel.

The patient was a girl, æt. 18 years, family history good. Duration of illness five weeks. This in the earlier stages closely simulated typhoid fever, and was later characterized by uncontrolled vomiting, diarrhœa, and irregular fever. At the autopsy, twenty-four hours after death, the lungs were found to be somewhat collapsed, but not tubercular; the heart normal; the abdominal viscera universally adherent; the omentum formed a thickened, broad band stretching across the epigastrium, which might have been detected before death; the entire peritoneum in both its visceral and parietal layers was studded with flattened and slightly umbilicated tubercular growths. The condition of the omentum, had it occurred in an elderly person, associated as it was with persistent vomiting, might readily have been mistaken for malignant disease of the stomach. *June* 28, 1888.

35. *Abscess of liver.*

Exhibited by Dr. T. R. Neilson.

J. W., æt. 37 years, single, a wheelwright by trade, was admitted to the Episcopal Hospital on September 3d. At that time he stated that he had enjoyed his usual fair health up to the middle of August, about three weeks previous to his admission.

For the last fifteen years was accustomed to drink about twelve large glasses of beer daily—did not drink whiskey or any other liquors. Apart from this, was a man of good habits. There was no history of any venereal trouble.

Family history showed nothing to account for his disease—all members healthy.

Patient worked steadily up to a month before admission. He had no previous illness except typhoid fever, which he had when a boy, and from which his convalescence was very slow.

About the middle of August began to feel decidedly unwell—began to lose strength and flesh rapidly. He suffered also from a slight pain in right hypochondriac region; and had chills, very irregular in character, and followed by very profuse sweating. Headache, anorexia, and wandering pains also were complained of. The bowels began to get loose—the stools being rather large and liquid—somewhat resembling those of typhoid fever.

When admitted, three weeks after the symptom began, he very much resembled a patient suffering from typhoid fever. Tongue dry and coated; face somewhat flushed; abdomen tympanitic. Temperature 103° F., pulse 112, respiration 30. On the third day after admission patient had a severe chill, lasting about an hour, and followed by profuse sweating; this was followed by another two days afterward.

An examination of the abdomen upon the day of admission revealed an enlarged liver—the dulness extending to median line and to three fingers' breadth below ribs anteriorly. There was a tumor also below the external surface in the right hypochondriac region. Over this tumefaction fluctuation could be detected, and friction was audible to the ear and perceptible to the touch.

This swelling increased steadily; the liver dulness extended to the left and downward also.

After admission the stools became frequent, large, and watery, but not free from bile. There was some tympanites and some passive congestion of the lungs.

On the 15th of September the abscess was aspirated, and, pus being found, the patient was etherized and an incision made in the right hypochondriac region $2\frac{1}{2}$ inches in length, at a distance of $2\frac{3}{4}$ inches from the median line. The peritoneum was stitched to the liver and a drain-tube put in cavity. The abscess was evacuated.

At the time of operation the tumor extended from the xiphoid cartilage to within one inch of umbilicus.

For eighteen hours after the operation the discharge from the drain-tube was very free—consisting of pus and some blood.

After the operation the lungs became more congested; cough and expectoration increased. The bowels remained troublesome, and the tympanites unrelieved. The temperature remained comparatively low until the evening following the operation, when it rose to 102.5° F., followed by a morning fall. Death took place at 12 P. M. Monday, September 17th, about sixty hours after the operation was performed.

September 27, 1888.

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36. *Typhoid fever.*

Exhibited by Dr. J. H. Musser.

I submit these specimens simply to show how aggravated a case of typhoid fever may be with a limited amount of local intestinal lesion. These intestines, as you see, contain but two or three ulcerated patches, and a few ulcerated solitary glands. The other patches are scarcely to be found on account of the small amount of infiltration. The mesenteric glands are swollen, dark-red in color, and present central softening.

The patient, a female, æt. 24 years, was admitted on the seventh day of her illness into the Presbyterian Hospital, with all the symptoms of typhoid fever. Her temperature ranged from this day until death—or the thirteenth day of the disease—from 103° to 104° F., presenting no marked falls in the twenty-four hours. The pulse was not excessively rapid—the frequency never getting above 110 until the last day of the disease; the respiration ranged from 30 to 32 until the last day, when it reached 42. A well-marked regurgitation murmur was detected

during life, but no organic defection complicating the disease. The diarrhœa was not excessive nor the tympanites marked. She was wakeful, however, but not delirious until three days prior to her death. The delirium became very wild in the last twenty-four hours, terminating in stupor, coma, and death. Apart from the active mental symptoms there were no grave phenomena, and at the autopsy, save the lesions just noted, nothing was found. *October* 11, 1888.

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37. *Typhoid fever, with epididymitis.*

Exhibited by Dr. J. H. Musser.

B. N., æt. 16 years, white, sailor, who said he had never had gonorrhœa, or even had intercourse with a woman, and who had never taken stimulants—a remarkable history for a sailor—was admitted to the medical wards of the Philadelphia Hospital, September 9, 1888, complaining of pain in the left testicle, also pain and tenderness in the right inguinal region. The pain in the testicle was accompanied with nausea. His temperature on admission was 103° F., pulse 120. The testicle was enlarged, very firm, and extremely sensitive. There was also some swelling along the spermatic cord. He stated that he attributed this inflammation to strain on the second of the month while wheeling heavy barrels of coal. During the week succeeding this—and hence the week prior to admission—he also had diarrhœa. There was no free discharge from the penis on admission, although one or two suspicious drops were observed twice at the meatus. There was nothing peculiar otherwise in the patient, and it was thought by one of my colleagues that ordinary gonorrhœal orchitis was present. He was transferred to the venereal ward. In the first week, although the patient had diarrhœa, the local inflammatory symptoms were the most marked. On the 20th of the month he was returned to my ward, and then well-marked symptoms of typhoid fever were present. Temperature from now until his death ranged from 102° to 104° F., pulse 110 to 120, subsultus tendinum, severe diarrhœa, extreme restlessness, with dry, brown tongue, and sordes on the teeth, were marked. His spleen was enlarged and some spots were found on the abdomen. The tenderness of the testicle continued, the organ and the cord to the inguinal canal were much swollen. The last week the testicle became soft. Exhaustion became extreme, and he died at 10 o'clock of heart-clot.

At the autopsy the usual lesions of typhoid fever, in the third week of the disease, were found. The omentum was attached at the internal ring by an old adhesion. The canal was filled with pus. Along the cord to the abdomen an enormous amount of it was found. The epididymis and the testicle were the seat of acute inflammation, with softening, and in both of which and in the tunica vaginalis testes pus was found. The old adhesion prevented the pus leaking into the peritoneal cavity.

Dr. Shakespeare studied the pus carefully, and found by microscopical examination and by cultures the presence of Eberth's bacillus, and particularly noted the absence of the gonococcus.

The case is of interest on account of the occurrence early in the disease of this rare complication, and of the difficulty to distinguish it by ordinary means from gonorrhœal orchitis. *October* 11, 1888.

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38. *Two cases of perforation of the vermiform appendix due to foreign bodies.*

Exhibited by Dr. H. F. Formad.

Perforation of the vermiform appendix is not rare, for I have in my collection of pathological specimens examples of thirteen cases. Perforation due to foreign bodies is however much rarer, for in only three of these cases was a foreign body found. Two of them are the cases shown to-night, the third case was one in which a prune-stone was found free in the peritoneal cavity.

Case I. From the body of a girl, æt. 17 years, who while in good health was seized suddenly with excruciating abdominal pains. She died on the fourth day. At the autopsy four cherry-stones were found, one in the appendix, three free in the peritoneal cavity.

Case II. Has a history very similar. In the appendix opposite the point of perforation was found a piece of raw chestnut.

October 25, 1888.

39. *Deformity of liver simulating movable kidney.*

Exhibited by Dr. J. H. Musser.

The liver which I show you was removed from the body of a woman, æt. 57 years, who died of consumption in my wards of the Philadelphia Hospital. During life a movable mass was felt in the transverse umbilical line midway between the ribs and the anterior spinous process. Its anterior edge was in apposition at this point with the abdominal wall, and was rounded, firm, and tender on pressure, creating some nausea. Its posterior border could not be defined, but shaded off into the loin. At times the mass could be more readily defined, and with the hand pressed firmly against the right loin—as usual in kidney palpation—the mass could be pushed forward, moving as far anteriorly as the median line, the edges then becoming well defined, rounded, and its shape apparently reniform, with its convex surface toward the ilium. The liver dulness extended to one inch below the margin of the ribs. Resonance anterior separated the liver from the outgrowth below, and hence it seemed apart from the liver. Moreover, it did not move synchronously with respiratory movement because the lower part was close to the ilium. Its movability led us to think, particularly as it appeared to be separated from the liver, that the tumor was a movable kidney.

In this specimen you will see that the elongated posterior third of the right lobe of the liver was the cause of the apparent renal tumor. When the patient was quiet the mass lay chiefly posteriorly in the loin. The anterior border or edge of the elongated portion was parallel to the nipple-line, on a lower plane, extending, as seen at the autopsy, from two inches below the margin of the ribs corresponding to the attachment of the liver, to the crest of the ilium. On pushing the mass from behind, this elongated portion revolved on the main part of the lobe, and the lower border, which lay in the iliac fossa, would be brought forward to the spinal column, producing the convex surface, posterior, like the kidney, while the concave corresponded, in a measure, to the hilus of that organ.

In connection with this specimen, I would call attention to the specimens removed from the body of a patient æt. 91 years, which were exhibited at the last meeting. In that instance we had examples of lobulation of the liver due to transverse atrophy on account of pressure, and, therefore, pendent from the right and left lobe, respectively, were two masses perfectly free to move in the abdominal cavity.

In these two instances we have a representation of tumors due simply to deformity of the liver. I recall but one instance only in a large number of autopsies of elongation of the right lobe of the liver to an extent comparing with this. In that instance, however, instead of the posterior portion of the lobe of the liver, the anterior portion of the lobe was extended and could readily be determined to be connected with the liver. (See *Pathological Society Transactions*, 1880.)

November 8, 1888.

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40. *Hydatid cyst of the liver.*

Exhibited by Dr. J. H. Musser.

The patient was an old woman who died of pneumonia. The appearances of the walls of the cyst indicate its progress to natural recovery. The walls were thickened and corrugated, and their outer surface distinctly marked off from the hepatic structure. A few daughter-cysts are seen with clear fluid contents, and, on microscopical examination, the echinococcus hooklets and cholesterine plates were found. It was seated in the lower portion of the right lobe of the liver, and is the size of a lemon. *November* 8, 1888.

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41. *Cancer of the stomach; extreme mobility of tumor.*

Exhibited by Dr. Osler.

J. G., æt. 65 years, admitted into Philadelphia Hospital Oct. 14th. Supposed to be suffering from malaria. Had a bad attack of chills and fever in the spring—has had occasional chills through the summer. Has lost flesh for some months. During the first three weeks of his stay in the hospital, he had two chills, in which, however, the temperature did not rise very high. The blood was examined with negative results.

It was not until early in November that he complained particularly of gastric symptoms. On inquiry it was ascertained that for some weeks he had had distress after eating, and on several occasions had vomited his food.

November 11th the following note was made: Patient is anæmic and emaciated; the abdomen flattened. There is a prominent projection below the left costal border in para-sternal line, reaching nearly to the navel and descending with inspiration.

Palpation reveals a firm, hard mass occupying the left hypochondriac region and the left half of the epigastric region. It is smooth and not painful, and can be moved from side to side to an extent of two or three inches. On percussion there is a flat tympanitic note. Liver dulness not increased. Glands in the groin are double the normal size. Vomited matters are brownish in color, acid, no sarcinæ.

The tumor changed curiously in position from day to day. At one time it would be far over in the right hypochondriac region entirely beyond the middle line, but more commonly a greater portion of its extent was in the left hypochondriac region. On several occasions it seemed to have disappeared altogether, and only a hard, small mass could be felt far over in the left hypochondria. Patient sank gradually, and died November 20th.

When the abdomen was opened, no trace of tumor was visible until the stomach was pulled down and to the right; it was then seen that the mass had fallen back into the left hypochondria below the ribs, where it was completely covered by the splenic flexure of the colon. The duodenum was curiously elongated and straightened; from the pyloric ring it measured over two inches as a straight tube. The pancreas was also drawn over to the left. The tumor could readily be pushed to occupy the positions in which it was felt during life. It involved the anterior wall of the stomach, which, when opened, presented a large hemispherical mass involving three-fourths of the circumference of the pyloric region, and extending to within an inch and a half of the ring. The surface of the mass was ulcerated, and at the base near the greater curvature suppuration had taken place. The pyloric ring itself, though uninvolved in the larger mass, was thickened and infiltrated.

There were no secondary growths in the other organs. Death had resulted from a low pneumonia in the middle lobe of the right lung, which was in a state of hepatization.

Dr. Osler remarked that this case was interesting on account of the unusual mobility of tumor and its extreme variability at different examinations. The position undoubtedly depended very much on the condition of the stomach—whether full or empty.

November 22, 1888.

42. *Case of carcinoma (medullary?) of œsophagus and cardiac end of stomach, with perforation of stomach.*

Exhibited by Dr. H. M. Fisher.

H. B., æt. 68 years, widower, portrait painter, was admitted to the Episcopal Hospital, October 25, 1888. During his stay in the hospital, patient was able to take nothing but small amounts of liquid food, and for a week before his death the ingestion of milk even caused so much pain that he refused almost all nourishment, and emaciation became extreme. A hard, nodulated mass could be distinctly felt in the epigastrium. He died November 17th. Post-mortem examination revealed a mass of medullary carcinoma involving the lower end of the œsophagus and the cardiac end of the stomach. The walls of the stomach had been completely ulcerated through over a surface about one and a half inches in diameter near the cardiac end, and there were also two smaller perforations.

The pancreas appeared also to have been involved in the degeneration.

The spleen was atrophied and showed numerous hard, white nodules upon its upper surface (capsulitis). The heart was very small and contracted. Coronary arteries intensely rigid and calcareous, and pericardium adherent. *November* 22, 1888.

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43. *Suppurative hepatitis (abscess of the liver).*

Exhibited by Dr. J. C. Wilson.

J. F., æt. 28 years, Italian, laborer; admitted to the Men's Medical Ward of the Jefferson Hospital the 26th of October, 1888. Patient had good health until May 30, 1888, when, while sleeping, he fell from a second-story window and received an injury of the back in the lumbar region; suffered from considerable pain, but in a week was able to resume his work; has had, however, a good deal of pain in the small of the back, more severe on the right side, from that date, and has not been able to stand erect on account of this pain; these pains, moderate at first, gradually increased. At the same time his general health became impaired, and on October 1st he was obliged to quit work; at that date he began to suffer from a constant pain in the right hypochondriac region;

this pain was dull and heavy, with frequent exacerbations; it was lancinating in character, shooting across into the epigastrium. About October 15th, noticed a small tumor in the epigastrium, which rapidly increased in size until his admission into the hospital, when it was about three and one-half inches in diameter, circular, and rising above the surface to the extent of about one inch; has suffered recently from diarrhœa; upon admission patient was dull, apathetic, complained of constant pain in the right side extending from the lumbar region to the epigastrium, tongue brown and furred, no appetite, no vomiting, no history of chills or of chilly sensations; stated that prior to admission his temperature was 104°; upon admission, both the morning and evening temperature were about normal; heart-sounds feeble and sharp, no murmur; area of splenic dulness not enlarged; liver dulness extends from the level of the fourth rib one hand's breadth below the arch of the ribs in the mammary line, and is continuous with the percussion dulness over the above-mentioned tumor, which occupies the epigastric region below the tip of the ensiform cartilage; the abdomen is slightly distended; abdominal walls not œdematous; considerable œdema of feet, legs, and thighs; there is slight jaundice; urine is acid, amber-colored, of sp. gr. 1020, contains a small amount of albumin and traces of sugar.

October 28. The tumor in the epigastrium fluctuated; it was aspirated by Dr. D. D. Stewart, and three pints of a dark, chocolate-colored, purulent fluid withdrawn. Under the microscope, this fluid showed blood and pus corpuscles. The same day he vomited a large amount of fluid containing material like coffee-grounds, which showed, upon microscopical examination, blood corpuscles and sarcinæ ventriculi. The day after the aspiration, he had frequent loose movements of the bowels and passed large quantities of blood. From this time he gradually sank, and died, comatose, the 2d of November. His friends objected to a post-mortem examination; the liver was removed through a small incision in the epigastric region, and was found to be the seat of an enormous abscess occupying the greater portion of the right lobe; no other organs were examined. *November* 22, 1888.

44. *Suppurative hepatitis (abscess of the liver).*

Exhibited by Dr. J. C. Wilson.

P. McC., æt. 41 years, Irish; married; laborer; admitted to Philadelphia Hospital, ward 8, bed 23, on June 23, 1888. Family history good; both parents reached old age; one brother died of consumption; patient's health has been in general good; no history of venereal disease; habits intemperate until about eight months ago, since which period he has not indulged in drink. Mental condition dull; patient declined to answer questions; history obtained with difficulty; it appears, however, that for some weeks patient has suffered from looseness of the bowels. Present illness is said to have begun five days before his admission to the hospital, with nausea and vomiting, and severe pain in the epigastric and right hypochondriac region. Upon repeated questioning patient states that he has not had chills. On admission to the ward he walked with great difficulty, and appeared to be very ill; skin muddy; conjunctivæ not yellow; pupils unequal, left being larger than the right; both react to light; tongue small, red, and coated. He states that his bowels have been regularly moved once a day since his present sickness began, namely, five days ago. Temperature 101.4° F., respiration rapid and irregular, about 34, pulse 100. Complains of constant pain in right hypochondrium, extending up to the right shoulder; heart and lungs normal; no friction sounds to be detected in any part of the chest, not even upon most careful auscultation at the right base; liver dulness extends about a finger's breadth below arch of ribs in mammary line; upper border of liver dulness cannot well be made out on account of the pain and tenderness; area of splenic dulness not enlarged.

On the following day a few fine friction sounds were heard at the base of the right chest posteriorly. The urine was reported by the resident physician to be normal.

Patient was treated with small doses of calomel, with morphine p. r. n. A blister, 6 by 4, was applied to the right hypochondriac region. Before the calomel had time to act diarrhœa set in, the passages, numbering from three to four in twenty-four hours, being thin, and of a dark color.

June 27. Patient passed a small quantity of bright fluid blood in the

morning, and in the evening had four passages, each of which contained a large quantity of dark, clotted blood. Some traces of blood were present in the stools of the following day.

June 29. The blood disappeared, and did not recur. The temperature, which had ranged from 101° or 102 F°., A. M., to 103° or 103.5° F., P. M., reached 104.6° F., P. M.

July 1. Considerable bulging in right hypochondrium with œdema, which extends up to the nipple-line. No fluctuation. Upper border of liver dulness reaches the level of the nipple, its lower border extends two finger's-breadth beyond the costal margin. Anteriorly absence of respiratory murmur in the lower part of the chest, with diminished vocal resonance and vocal fremitus. At the angle of the scapula, posteriorly on the right side, there is a fine friction.

Diagnosis: Abscess of right lobe of liver pushing up diaphragm. A large aspirating needle was introduced in the right infra-axillary space, between the seventh and eighth ribs; about an ounce of dark-colored fluid pus was discharged; the needle moved freely in all directions, making it quite evident that it had entered a large abscess cavity, the contents of which, however, it was impossible to evacuate by this means.

July 3. The abscess was freely opened by Dr. Ransley, an incision two inches in length being made between the eighth and ninth ribs, in the line of the anterior axillary fold. A large amount of foul-smelling, thick, chocolate-colored pus was discharged. This fluid could not be measured, but was estimated to be about two pints. A drainage-tube was introduced, and the wound treated antiseptically. The operation was followed by a fall in temperature from 104° to 100° F.; the patient's general condition did not, however, improve; diarrhœa continued, the stools, numbering from ten to twelve in twenty-four hours, being dark and watery, but not containing blood. Constant nausea and vomiting rendered the administration of food and medicine alike impracticable. The pus discharged from the wound continued to be dark-colored, foul, and profuse. The mental condition was soporose, with occasional wandering delirium.

9th. The temperature has been gradually falling for the last forty-eight hours. Death occurred at 5 P. M., the temperature being at that time 97.4° F.

One of the more noticeable phenomena of the case has been the rapid emaciation. During the six days after the operation the patient's tissues appeared to melt away from day to day.

No systematic post-mortem examination of the body was permitted but with great difficulty an arrangement was effected by which a hasty examination of some of the organs could be made. The liver was large and fatty, gall-bladder distended, upper part of right lobe shows an extensive cavity, with irregular, shreddy, gangrenous walls, which form the anterior and lateral boundaries of an abscess the size of a cocoanut, the superior boundaries of which were formed by the diaphragm. This cavity, which appears to have been nearly globular, has a diameter of about seven inches. It is abruptly bounded by unchanged liver tissue. On the upper surface of the left lobe there is a small superficial abscess, about two inches in diameter and an inch in depth, with dark-colored, purulent contents. The stomach was of moderate size, and shows no lesions. Spleen normal in size and consistence. Kidneys showed no coarse lesions. No examination of the intestines could be made. There was a small, circumscribed pleural effusion on the right side, the pleural surfaces being elsewhere adherent by reason of inflammation; no adhesions on left side. Lungs free from tubercle, and crepitant throughout. No thrombi in branches of pulmonary artery; no infarcts. *November* 22, 1888.

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45. *Strangulated omental hernia.*

Exhibited by Dr. J. B. Deaver.

W. S., æt. 25 years, was admitted to my wards in the German Hospital on the evening of November 26, 1888, suffering from strangulated omental hernia, which occurred on Friday, November 23, 1888 (three days before admission to the hospital), by lifting a heavy weight. He stated that it came on suddenly, and that he had not had a hernia before this accident.

When admitted the patient was in a state of collapse, with a temperature of 96°, and a feeble and rapid pulse. Had a bowel action the day of accident, and not again until the day of admission to hospital, when he had several thin passages.

The swelling was tense, painful, and red, and did not give succussion. The patient had vomited several times since the day after the accident, which, when examined, proved to be only bilious matter. He was troubled constantly with hiccough. To my mind the subjective symp-

toms presented here, together with the presence of an irreducible tumor at one of the hernial orifices, which tumor had made its appearance suddenly, were those of a strangulated hernia.

Notwithstanding the looseness of the bowels, which was a symptom in this case, I felt convinced that the trouble was all attributable to the hernia and particularly so as the patient was so depressed. It is true that these symptoms could have been caused by a gastro-enteritis, yet I do not think there is any surgeon who would not have advised the course which I pursued, namely, to explore and be governed by the result of their exploration. I have seen cases of acute adenitis, particularly of the glands in the cribriform fascia, where the patients gave the history of the tumor having appeared suddenly, and following straining efforts, also where there were sick stomach and constipation. These are cases which simulate hernia, and where it may be impossible upon our first visit to make a positive diagnosis, and where, I think, other things being equal, it is equally advisable to explore, and if found to be a glandular swelling to remove it. I have recently seen a case of complete strangulated femoral hernia where the patient had several loose passages from the bowels after the occurrence of the strangulation, therefore I do not think that we should always expect to meet with absolute constipation. Again, it is better to operate under all circumstances, as it gives the patient the benefit of any doubt, at the same time gives the surgeon an opportunity to perform the radical cure, if it is a hernia, and thereby prevent the return of the hernia, in a large majority of cases, at least.

An incision was made over the line of the tumor, which included the upper part of the scrotum, and the different layers of tissue divided until the sac was reached. Upon opening the sac, not having attempted to reduce the mass before so doing, it was found to consist of the great omentum alone, and including nearly all of it, as the transverse colon presented at the orifice of the sac. The lower part of the omental mass was in contact with the testicle, and separated from the upper portion of the mass by a partition extending across the tunica vaginalis. In other words, this was a case of congenital hernia into the funicular portion of the tunica vaginalis; but the septum, where occlusion of the tunica had taken place, had given away in advance of the mass of omentum, allowing a portion of it to escape through it, thereby coming in contact with the testicle. This being a variety of congenital hernia other than the encysted form, or the infantile hernia of Hey, of Leeds, thus explaining its sudden appearance.

The omentum which I present this evening, having been in a questionable state, I tied it off with the catgut ligatures, and cut it away.

My experience has taught me that where the omentum has been out for any length of time, and where it is adherent to the sac, whether it has been strangulated or not, that it is best to treat it in the way that I treated it in this case. In cases of irreducible omental hernia, which I have met with, where the omentum was found adherent to the sac, also where a knuckle of gut had come down and become suddenly strangulated, thus calling for operation, having separated the omentum from the sac and replaced it, at the autopsy I have found it gangrenous; while, on the other hand, I have had no ill effects from cutting it away. Hence my reasons for this form of treatment. My object in not attempting the reduction of the hernia upon exposing the sac is two-fold, namely: that I think it safer to open the sac and expose its contents, and reposit them, for the following reasons: First, to determine their condition, as to whether they are sufficiently healthy to be replaced; secondly, to free them from the sac, thereby preventing a continuance of the symptoms having called for operation, and which would call for reoperation. I have known this to occur in the hands of other surgeons, where they have performed the first operation, and not having relieved the symptoms, had to come back and perform a second operation, when, upon opening the sac, it was found that the intestine was strangulated by an adherent band; thirdly, in the event of their not being in a condition to return, to enable us then either to resect the intestine or make a temporary or permanent artificial anus. Again, that it gives an opportunity to do Barker's operation, which I think is the rational one for the radical cure. I hold that the radical cure should be done in all cases of strangulated hernia particularly, unless the patient's condition at this stage of the operationis so critical as not to justify it. I not only do this operation in cases of strangulated hernia, but in those of reducible hernia, it being a perfectly safe and reliable one in the great majority of cases.

In addition to having performed a radical cure in this case, I cut away the portion of the sac intervening between the neck and the septum in the tunica vaginalis, closing the remaining portion of the tunic with a continuous catgut suture, thus making a complete tunica vaginalis for the testicle. The patient has progressed favorably, his temperature never having risen above 99°. I have dressed this case but twice since the operation, finding it entirely healed at the second dressing. The patient has required no opium, a drug that was formerly given after all operations of this kind. Other than a stimulant and tonic treatment, there have been no medicines given with the exception of a few

doses of sal rochelle, the second day after the operation, when the abdomen was slightly tympanitic, the result of the distended condition of the bowels.

In cases of old, irreducible omental hernia, complicating a recently-strangulated gut, where I have separated an adherent omentum from the sac in exploring the gut, after having relieved the strangulated bowel, I have reduced the omentum, and, to my disgust at the autopsy, I have found this, which appeared to me to be healthy omentum, at the time of the operation, to be gangrenous; while on the other hand I have had no ill effects following when having tied it off and cut it away.

December 13, 1888.

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46. *Acute yellow atrophy of the liver occurring after criminal abortion.*

Exhibited by Dr. H. F. Formad.

In the evening the woman was happy and well; the next morning she was seized with severe pains and died in two days, deeply jaundiced.

Autopsy, twelve hours after death, showed all the organs in a stage of advanced decomposition. The womb was badly lacerated. There was gangrenous endometritis, pyosalpinx, and severe peritonitis. The liver showed acute yellow atrophy, which had not reached an advanced stage. Under the microscope the cells were all degenerated, and there were abundant crystals of leucin and tyrosin. *January* 10, 1889.

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47. *Syphilitic cirrhosis of the liver.*

Exhibited by Dr. H. F. Formad.

The liver was in an advanced state of cirrhosis, and contained gummata scattered over its surface. It had been obtained from the body of a peddler who had plied his trade till within twelve hours of his death, never complaining of pain, and only occasionally of slight discomfort. He was found dead. The case is remarkable on account of the enormous peritonitis present. The peritonitis is not tubercular; there is no enlargement of lymphatic glands. Over the whole of the peritoneal surface is spread a thick layer of false membrane. The abdominal organs are matted together in one solid mass and the stomach

encroached upon. The kidneys are cirrhotic, and, as is the rule in syphilitic cases, one is much more advanced in the process than the other. *February* 14, 1889.

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48. *Ulcer of the stomach.*

Exhibited by Dr. J. H. Musser.

The ulcer, situated on the greater curvature, was produced by a submucous caseating tubercular deposit breaking through the mucous membrane. The edges of the ulcer were bevelled, but not thickened, its floor honeycombed and covered with a mass of purulent detritus. Extending around the ulcer was an area of thickening. Toward the cardiac end of the stomach were two nodules situated beneath the mucous membrane, which, on section, were found to consist of caseating tubercle. The patient had general tuberculosis. There had been no gastric symptoms. One of the supra-renal bodies was the seat of tubercular disease, but there was no bronzing. *February* 14, 1889,

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49. *Cancer of the pylorus.*

Exhibited by Dr. Downs.

D. D., æt. 46 years, white, Irish; occupation, circus-driver. Had usual diseases of childhood. Had chancre when 16 years of age, and suffered from occasional attacks of rheumatism. During the war, was shot in the mouth, the shot tearing away the alveolar process of the right supra-maxillary bone and the teeth of the left supra-maxillary. Has suffered from flatulent dyspepsia during past fifteen years; attacks of vomiting more or less frequent for the past year, the present attacks of vomiting occurring about every sixteen hours, without any relation to time of eating; vomited matter consists first of partly-digested contents of stomach and then thin, watery mucus. Has never vomited blood. No pain in abdomen, except just a short time previous to an attack of vomiting; always constipated. Last winter began to lose weight; actual loss now 65 lbs.

Post-mortem, twenty hours after death, showed a well-marked cancerous infiltration of the pylorus. *February* 14, 1889.

50. *Malignant disease of stomach (diffuse secondary growths in abdominal organs).*

Exhibited by Dr. CALLENDER for Dr. J. C. WILSON.

C. G., æt. 32 years, a groom, born in England, was admitted to the Jefferson Hospital, 25th of October, 1888. No hereditary predisposition, so far as can be ascertained, to cancer; general health good until last April, when he began to suffer from disturbances of digestion, heartburn, acidity, and severe lancinating pains in the epigastrium; a month later vomiting occurred, and has continued almost daily, and frequently several times a day, till his admission; vomiting almost invariably follows the taking of food; vomited matter was at first streaked with blood; through the summer, however, it frequently contained considerable amounts of bright blood and material looking like coffee-grounds; large quantities of mucus, and occasionally blood, were voided by the stools; since July, however, constipation has been noted with whitish, clay-colored stools; the urine has been somewhat diminished in amount, of acid reaction, normal appearance, without sediment, and has at no time contained albumin; noticed in June, for the first time, a circumscribed swelling in the epigastrium; this tumor was sensitive to pressure and rapidly increased in size, extending into the right hypochondrium; the liver was much enlarged, extending from the fourth intercostal space downward a hand's breadth below the costal border in the mammillary line; the area of splenic dulness was not increased; there is general abdominal tenderness, but neither tumor nor ascites can be detected; the legs and feet are not œdematous; inguinal lymphatics are enlarged on both sides; heart and lungs normal. The patient's skin is sub-icteroid; he is much emaciated; very weak and apathetic; temperature normal. Death occurred on the 19th of November, having been preceded by a gradual fall in temperature, which reached 93.3° F. on the day preceding dissolution.

Upon *post-mortem* examination the primary lesion was found to be an ulcerated growth near the pylorus, the lumen of which was, however, not interfered with. There was secondary implication of the liver, spleen, omentum, the inguinal and mesenteric glands, the muscles of the abdomen and the lower lobe of the right lung, the pancreas remaining free. The liver weighed 9¾ lbs., and showed on its surface and in its substance great numbers of globular masses of light color and medullary consistence, varying in size from a pea to an orange, many of those upon

the surface being dimpled. The heart and pericardium, the lungs, with the exception of a few scattered nodules on the lower lobe of the right lung, the pleura, and the kidneys were normal.

Upon microscopic examination the primary growth was found to be glandular carcinoma. *February* 28, 1889.

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51. *Intussusception of ileum and large intestine with meagre symptoms.*

Exhibited by Dr. M. H. Fussell.

J. S., æt. 7 months, was ill with diarrhœa for several days previous to Monday, February 18, 1889. Early in the morning of that day he was seized with vomiting accompanied by considerable retching, simply the breast milk was regurgitated however. During the morning he had a movement of the bowels consisting of a thin fecal matter and liquid blood; there was *no tenesmus*.

I saw him in the evening; vomiting was incessant, everything taken into the stomach was regurgitated now without any retching, however, and the vomited matter consisted wholly of the contents of the stomach.

There was no evidence of pain. The belly was soft and careful palpation failed to elicit any spots of tenderness or any tumor.

During the evening of the first day there was a second stool of thin fecal matter and liquid blood without pain or tenesmus. The child's countenance was pinched, the temperature normal.

The patient was fed exclusively on breast milk, and searching for a cause of the trouble, I found the mother had partaken of chicken the previous day that was unfit for food.

Her milk was examined and found to be of a *dark-green* color, and without odor, but evidently unfit for the child.

The patient was ordered cow's milk, barley water, and gruel to the exclusion of breast milk, and powders consisting of $\frac{1}{12}$ grain of calomel and 2 grains of bismuth were ordered every two hours.

Next morning vomiting still continued, but at lengthened intervals without pain. During the day there were three or four liquid fecal passages, free from blood and passed with *no pain* and *no* tenesmus.

During Wednesday night the child slept almost all the time. Thursday there was but little vomiting, no pain, no tenesmus, and several liquid, ill-smelling, fecal passages. The stools were so free from pain that the mother would not be aware that the diaper was soiled until she detected the odor of the passage.

I was called at 3 A. M. on Friday and found the child in evident pain for the first time. The belly was again examined and found soft. No evidence of tumor, owing doubtless to the thick belly walls. The child had vomited but once since 9 P. M. on Thursday, there had been several ill-smelling stools during the night still passed without pain and free from blood. I recognized that the child was in a serious condition but was unable to satisfy myself as to the cause of the evident collapse. He died at 7 A. M. At the post-mortem the belly walls were found exceedingly thick, owing to a layer of fat. Belly not distended. Thoracic organs were normal. Intestines but slightly distended and not markedly congested. No signs of peritonitis.

On searching for the colon it was found to be absent from its normal position, and the cæcum, ascending colon, and transverse colon were found in the region of the descending colon.

The tumor was not at all congested and looked like a rather puckered large intestine filled with feces. There was a marked absence of the resemblance to a large blood-clot as described by Smith.

On dissecting the parts the tumor was found to be six inches long, extending from about one and a half inches above the anus upward into the descending colon.

The invaginated mass was found to consist for about two inches of its length of ileum, the rest consisting of the cæcum and colon. The outer layer was the greatly puckered colon. A large lead pencil could easily be passed through the lumen of the invaginated gut.

The ileum was much congested and the extreme lower portion quite œdematous.

The gut above the constriction was but little congested. This is the first case of invagination of the intestine which I know to have occurred in my practice, but I am confident that I have given many certificates for entero-colitis where there were just as positive signs of invagination as in this case.

Smith says that liquid blood passed unmixed with fecal matter is considered by some as pathognomic of intussusception. Here, during the first twenty-four hours the patient was under observation, there were two bloody stools, but both movements consisted partly of fecal matter. The following passages consisted of ill-smelling, watery stools passed without pain or tenesmus, and free from blood. The vomiting became less as the case progressed, it was always mild, and never the least approach to a stercoraceous vomiting. There was no evidence of pain until four hours before death. There was never any tympanites; never any tenderness of the abdomen.

In the light of these symptoms I considered the case one of entero-colitis, due to the mother's deranged breast milk, and so considered it until I was enlightened by the post-mortem.

No one could have been more surprised than I was when I saw the lesion above described, and I was forcibly reminded of Prof. Osler's remark, "that we never know what is inside our patients until we have them on the *post-mortem* table."

I presume that the meagre symptoms in this case were due to the fact that the ileo-cæcal valve was abnormally large, and allowed this great amount of invagination without causing obstruction to any marked extent. *February* 28, 1889.

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52. *A case of tuberculosis with unusually large deposits in the liver.*

Exhibited by Dr. A. V. Meigs.

The patient was a mulatto, æt. 26 years, and a waiter by occupation. He could give no account of his family history, but said he had always enjoyed good health, though subject to nose-bleeding. For eight days he had epistaxis four or five times a day, which at last became so severe and prolonged that he was much prostrated, and on September 25, 1888, came to the Pennsylvania Hospital. During the week previous to his admission he had slight cough but no expectoration. There had been neither diarrhœa nor constipation—he had never had any venereal disease, nor had he been a hard drinker. He had, while in the hospital, continued fever of moderate degree, the temperature never exceeding 103°, and usually it varied between normal and 101°. No satisfactory diagnosis was ever made—the question of the existence of typhoid fever was debated but dismissed as improbable; then it was thought not unlikely pulmonary tuberculosis might exist, as physical examination made it evident there was lung disease of considerable extent, but toward the end it was considered most likely there was amyloid disease of the liver as that organ seemed a good deal enlarged, whatever other disease might exist. After having had epistaxis again and again and several hemorrhages from the bowel, he died on October 21st, having been in the hospital four days less than a month. While in the hospital there was never any expectoration nor cough enough to attract attention, though careful questioning elicited that he did cough a little from time to time.

The autopsy revealed the following:

Heart natural in every respect except that the aortic opening was slightly narrow, and the aorta presented upon its inner surface a few yellowish patches.

Lungs: The left was markedly smaller than the right (two-thirds size), and both were extensively infiltrated with yellowish nodules, none of which were broken down or even softened. The left pleura was very thick and tough and the right exhibited the same change but in less degree.

Liver about one-third to one-half larger than natural. The capsule was very much thickened, especially upon the upper surface of the left lobe, and this thickened capsule was everywhere infiltrated with yellowish nodules as large as the end of a finger. The *liver* was everywhere infiltrated and its surface studded with nodules, some of which were as much as half an inch in diameter. Many of them had broken down and their centres were filled with a bile-stained liquid. The walls of the portal veins were much thickened and the vena cava also was thicker than natural.. On section the tissue creaked under the knife and the cut surface was perceived to be rough and granular when the finger was passed over it.

Spleen was of about natural size, but the capsule enormously thickened, and both it and the organ itself contained many masses similar in size and appearance to those in the liver.

Intestines were everywhere firmly matted together by adhesions which were thick and tough, and studded with tubercles, many of them as large as the end of the little finger.

There was a cavity in the right side of the pelvis which was filled with pus.

Kidneys were slightly enlarged and pale. Microscopical examination was made of the lungs, liver, spleen, pancreas, small intestine, and kidneys.

Sections of some of the masses in the liver presented externally a zone of liver cells, more or less flattened and exhibiting slight nuclear proliferation; next a circle of round-cell infiltration staining bright red with carmine; then a band of fibrous material containing a few scattered cells but pale in color; next a broad band bright red in color thongh less so than the outer circle, and containing a few cells but otherwise almost structureless in appearance. Within this was a fibrous looking material, not staining with carmine at all, but more or less discolored with bile pigment. This material filled the interior of the spheres except in such tubercles as were so large that they had softened

in the centre and were filled with a bile-stained fluid. The largest tubercle cut for microscopical examination measured three-eighths of an inch in its longest diameter. In the sections others are seen of all sizes down to the most minute points of round-cell infiltration. In places around the larger masses the capillaries are filled with red blood-corpuscles.

Some of the masses in the peritoneal layer of the small intestine are almost pedunculated and exhibit the same structural appearances as those in the liver. The largest one cut measured three-sixteenths of an inch in diameter. The submucous coat of the intestine is infiltrated and the mucosa much thickened as though hypertrophied.

The masses in the spleen are in no wise different in appearance from those already described, except that the outer zone of round-cell infiltration is less obvious owing to the nature of the tissue in which they lie.

The sections of lung present the usual appearances.

The pancreas exhibits unmistakable evidence of degeneration, there being at one place in the sections marked disintegration of the cells and—what is still more striking and obvious—in many positions whole lobules contain no secreting cells at all, but their places are taken by a loose-meshed connective-tissue reticulum containing lymphoid cells, but these not closely crowded together. The change seems a degenerative one.

Sections of the kidney present no tubercular deposits but marked parenchymatous degeneration.

A casual examination of some of the text-books of pathology shows that there is a good deal of difference of opinion in regard to the subject of tubercular disease of the liver. Wilks and Moxon (*Pathlogical Anatomy*, Philadelphia, 1875, p. 458) say, in speaking of tuberculosis in general, that there are two different forms, "the primary local tuberculous nodule or mass, which becomes caseous and softens, and the secondary shower of scattered minute grains, familiarly known as miliary tubercle. It is important to notice that the first kind, viz., primary and local tubercle, does not occur in the liver; at least we have never seen it. The older descriptions of such tubercles probably refer to syphilitic disease or certain forms of sarcoma." Rokitansky (*Pathological Anatomy*, "Sydenham Society Translations," vol. ii. p. 149) says, "contrary to the received opinion, we assert that the liver is rarely the seat of tubercular disease. It scarcely ever occurs in this organ as a primary affection, but is not unfrequently found as a secondary complication of advanced primary tuberculosis in another organ, or of uni-

versal tubercular disease. . . . . The tubercular matter is deposited in the parenchymatous cellular tissue of the organ, and especially in that pertaining to the biliary capillaries. It very frequently surrounds a minute gall-duct, and thus presents a central canal, which gives rise to a biliary discoloration of the nucleus." Rindfleisch (*Pathological Histology*, "New Sydenham Society Translations," vol. ii. pp. 119, 120) says "in general tuberculosis of the serous membranes, the lungs, the heart, etc., such as is most commonly met with in children, a variable number of miliary nodules may also be found scattered through the liver. They are developed upon the smallest ramifications of the hepatic artery. . . . . Even should the tubercles in the liver, as occasionally happens, attain very considerable dimensions, they nevertheless remain of very subordinate local importance." Cornil and Ranvier (*Pathological Histology*, translated by Shakespeare and Simes, Philadelphia, 1880, p. 559) make the following statement: "Tubercle granules are very common and often very numerous in the liver of patients who have succumbed to an acute miliary tuberculosis. . . . . The miliary granules in the substance of the liver are seated in the connective tissue which accompanies the portal vessels. . . . . They do not then differ from miliary tubercles elsewhere; they rarely exceed 1 to 10 millimetres in diameter. Those of the largest size are formed by an agglomeration of several smaller ones, and are caseous and soft." Ziegler (*Pathological Anatomy*, translated by MacAlister, London, 1884, Part II., Section viii. pp. 337, 338) expresses himself as follows: "Tuberculosis of the liver appears in two chief forms, namely, as miliary tuberculosis and as tuberculous hepatitis. . . . . In the second form of tuberculosis, or chronic tuberculous hepatitis, there is not only an eruption of nodules, but also a diffuse fibrous hyperplasia of the liver. . . . . Cases occur in which the entire organ is honeycombed with innumerable cavities, varying in size from that of a pea to that of a walnut; but such cases are rare." Dr. Morris Longstreth, who is the pathologist of the Pennsylvania Hospital, allows me to say that though he has quite often seen tubercles in the liver as large as those found in this case, he cannot remember ever to have seen them except in people belonging to the negro race.

Thus it becomes evident that in a common way, at least, the subject is not as well understood as is to be desired. No one of the authorities quoted alludes to a case seen, or gives a plate (and some of the books are profusely illustrated) exhibiting appearances parallel to those of the sections which are upon the table, and it is probable some one of them would have done so if the material had been at hand. It is curious

to note the differences of opinion expressed in regard to the situation in which tubercles in the liver originate and the size they may attain. My own view is that tubercular nodules so large as those I have exhibited, are decidedly uncommon in the liver, for I am free to confess that this is the first instance in which I have seen them, and further that the explanation of the opinion expressed by the English observers (Wilks and Moxon) is to be found in Dr. Longstreth's statement that he has seen them only in people belonging to the negro race. At Guy's Hospital, where they made their observations principally, but very few negroes can come to the autopsy table. One thing more and I shall have finished, the disease in this case was so much more extensive and further advanced in the liver than in any of the other organs, unless it be considered that the tuberculosis of the peritoneum was a more advanced process, that it is not unlikely the disease may have had its origin in the liver, and this is further borne out by the clinical history of repeated and increasing epistaxis, and enlargement of the liver, revealed by physical examination, though to be sure these symptoms may, perhaps, almost equally fairly be attributed to tuberculosis beginning in the peritoneum.

February 28, 1889.

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53. *Obstruction of the common bile-duct by a large calculus.*

Exhibited by Dr. A. A. Eshner.

Two days before admission to the hospital, on March 23, 1889, the patient was seized with a severe rigor, with nausea and vomiting, with pain in the abdomen, with jaundice and with anorexia. The chill was repeated on the following morning, and the other conditions continued. Patient made the statement that he had been in the habit of drinking large drafts of cold water, to which he ascribed his symptoms. On admission icterus was evident. The tongue was heavily coated and there was complete anorexia. The bowels had been moved once or more daily for some time. The stools examined contained masses of undigested milk curd. There was general abdominal tenderness. The temperature was 103.4°. Some increase in the hepatic dulness was detected. The lungs were congested. The urine contained a small quantity of albumin, chlorides, biliary coloring matters; no sugar and no casts. The temperature continued high, and there were repeated chills at irregular intervals. Under treatment by aconite, quinine, and

morphine, in a few days the temperature fell, but the jaundice grew steadily more intense. On about the seventh day hiccough set in, accompanied with the ejection of a small amount of blood-stained mucus. For a day preceding death a moderate amount of bloody fluid oozed from the patient's mouth, and immediately preceding death, on the eleventh day after the first chill, he vomited a considerable quantity which, however, was not measured. At the autopsy the tissues generally were found laden with an excessive amount of adipose tissue. The pericardium contained a slight excess of blood-stained fluid. The heart itself was normal. The stomach and intestines contained quite a large quantity of bloody fluid. The liver was large and weighed six pounds. In the left lobe was a fluctuating area which, on incision, gave exit to a dirty, yellowish fluid, and exposed the hepatic canals dilated into saccular cavities. Sections of the liver at numerous points permitted the exudation of fluid of the same kind, of which, however, there was but little in the right lobe. In this lobe, visible on the upper surface, was a small calcareous nodule of the size of a pea. The gall-bladder was small and contracted, of about the size of a walnut, and contained a brownish bile, together with small particles of a hard greenish material. The hepatic duct was greatly dilated. In the common bile-duct, just beyond its commencement, lay somewhat loosely, at any rate not tightly, a gall-stone of about the size of a hickory nut, oval in shape and apparently made up of the same material as the fragments found in the gall-bladder. On transverse section of the pancreatic duct a few drops of a yellowish, creamy fluid escaped in a jet. Numerous sections of the pancreas, however, showed no gross changes. The spleen was large and pulpy. The kidneys were both large and surrounded by a luxuriant layer of fat, with the removal of which the capsules also came away. One of the organs contained a small number of cysts. Both showed fatty changes in the parenchyma. *March* 14, 1889.

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54. *Cancer of gall-bladder.*

Exhibited by Dr. H. F. Formad.

The patient from whom this interesting and instructive specimen was obtained was a woman past middle age. Last July she was suddenly attacked with extremely severe vomiting and purging and excruciating abdominal pain. She almost died then, but got over the attack and

eventually returned to almost her normal health. In September, jaundice suddenly developed, the stomach became irritable, the bowels obstinately constipated. The jaundice persisted, she lost flesh steadily, and died a week ago. The gall-bladder was found much diminished in size—contracted from old, chronic inflammation. Its walls were irregularly thickened, and sections examined under the microscope showed this thickening to be due to cancerous deposit. About the gall-bladder the tissues were inextricably matted together, and it was evident that at one time a stone had ulcerated through into the intestine. It is probable that the cancer developed in the cicatrix due to this ulceration. In the gall-bladder were found twenty-five little stones, with no bile. The cystic duct was completely occluded. The liver was uninvolved in the cancerous degeneration, but was in a condition of acute yellow atrophy. Pancreas was normal. Kidneys were contracted and granular.

April 11, 1889.

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55. *Corrosive sublimate poisoning.*

Exhibited by Dr. H. F. Formad.

The mucous membrane of the stomach was in a state of intense congestion, evidently the result of some irritant poison, it was not known what. There was not the alligator-skin appearance of the mucous membrane so characteristic of arsenic poison. The appearance pointed strongly to corrosive sublimate. Another thing which made it look like corrosive sublimate was the manner of death. The man came home well, ate a hearty meal, put something into a glass and drank it, and was dead in fifteen minutes.

May 9, 1889.

III. THE VASCULAR SYSTEM.

1. *Thrombosis of portal vein.*

Exhibited by Dr. William Osler.

Chronic obstruction to the blood-flow in the branches of the vena portæ within the liver, such as occurs in cirrhosis, is probably the most common cause of this rare condition. We not unfrequently meet with atheroma in the portal vein and its branches in cirrhosis, one factor in the production of which, as in the pulmonary artery in mitral stenosis, is the heightened blood-pressure, and to this change the thrombus formation is closely related. At first, and possibly for a long time, mural, it gradually becomes obliterating, and if the collateral circulation is established the patient may live some time, as in a case which I reported in 1882 (*Journal of Anatomy and Physiology*), in which the vein was represented by a firm fibrous cord. The specimen here shown was taken from a Swede, æt. 45 years, who was admitted to the University Hospital on May 7th with ascites. As he could not speak English it was difficult to obtain a history, but we ascertained that he had been in failing health for some time, and latterly his abdomen had become swollen. It did not appear to have come on suddenly. The liver dulness was reduced, the superficial abdominal veins were moderately distinct, and the case was regarded as one of ordinary cirrhosis. He was tapped five times between May 20th and July 14th, and from twelve to fifteen pints of serous fluid removed on each occasion. He was able to be up and about after each tapping, but he gradually became emaciated, weak, finally comatose, and died on the 20th of July. There were no hemorrhages, and it may be noted that the abdominal veins were at no time more distended than is common in atrophic cirrhosis. The condition post-mortem was as follows: Fibrous adhesions in peritoneum; atrophic cirrhosis of liver, with thickened capsule; old, firm thrombus in vena portæ, extending into splenic and mesenteric veins; thickening and patchy calcification of the walls of the portal vessels; great enlargement and induration of the spleen; kidneys indurated; no special changes in thoracic organs. A careful dissection of the abdominal veins was not made, but I am told by Dr. Miall, who made the autopsy, that the peri-

toneum was very dark and the veins behind the liver very large. The collateral channels were, no doubt, largely the anastomoses which exist between the peritoneal, mesenteric, and lumbar veins, and gastric, diaphragmatic, and œsophageal vessels. As in the case of fibroid obliteration referred to, a perfect collateral circulation may be established in occlusion of the portal vein, and the patient may live for months or years. In this instance the thrombus was evidently old, as in places there were calcareous changes. The branches in the liver were filled with soft clots. The gall-bladder was full of normal bile.

In two other cases of cirrhosis I have met with thrombi in the portal vein, in both instances with thickening and atheromatous changes in the vessel-walls, but the thrombi had not undergone such degenerative changes as in this specimen. *September* 22, 1887.

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2. *Gunshot wound of heart.*

Exhibited by Dr. H. F. Formad.

The ball entered the chest between the fourth and fifth ribs on the left, passed through the left ventricle, and was found imbedded in the spinal column. Death was instantaneous. The principal point of interest lies in the fact that the ball traversed the cavity of the left ventricle near its base without injuring the chordæ tendineæ or the leaflets of the mitral valve. An explanation of this is evidently to be found in considering that the bullet, when it struck the heart, had lost much of its initial velocity, and, too, the amount of blood that happened to be in the heart at the moment it was struck predisposed to the slightest degree of injury to its internal structures. *September* 22, 1887.

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3. *Aneurism of aortic arch.*

Exhibited by Dr. E. T. Bruen.

Thomas F., æt. 42 years, was an inmate of the Philadelphia Hospital some six months prior to his death, which occurred September 30, 1887. He had been a syphilitic subject, and had suffered from dyspnœa and

cough; the increasing severity of these symptoms forced him to seek the hospital.

The autopsy exhibits two aneurismal dilatations of the aorta—one of these is situated on the superior surface of the ascending arch. It is as large as a small lemon, and partly filled with a laminated clot. The other aneurism projected from the inferior aspect of the commencement of the arch, near the origin of the common carotid and subclavian on the right side. The calibre of these vessels had not been influenced by the aneurismal tumor. The aortic valves were normal; the walls of the left ventricle slightly thickened, but not to a marked extent. The intima of the aorta beyond the aneurisms showed atheromatous changes. The upper lobe of the right lung had undergone atrophy and splenification from pressure. The bronchial glands were enlarged, but there were no other lesions of consequence.

The clinical history of this patient while in the hospital exhibited the ordinary pressure symptoms common to aneurismal tumors. There was compression of the upper lobe of the right lung with splenification of a portion of it. Difficulty in deglutition was pronounced, and markedly stridulous breathing. I have brought the specimen before you to emphasize two facts. The first of these is the extraordinary absence of any implication of the laryngeal muscles. The vocal cords moved freely and yet there must have been irritation of the recurrent laryngeal nerves. It is true that there was a barking cough characteristic of laryngeal irritation, but no spasm of the laryngeal muscles.

The second feature of interest is the button-hole narrowing of the distal portion of the aorta, just beyond the sac of the aneurism on the surface of the arch. The blood passing through this orifice created a murmur which had a diastolic rhythm. This murmur was audible near the second left costal cartilage, and was the occasion of some doubts as to the exact diagnosis. *November* 10, 1887.

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4. *Disease of the coronary arteries; fibroid heart.*

Exhibited by Dr. W. Osler.

The specimen illustrated an unusually widespread obliteration of the branches of the coronary artery in a comparatively young man, who had never shown any symptoms of heart disease. The man, aged about 35 years, had been an inmate of the Pennsylvania Institution for Feeble-

minded for eighteen years. He had been brought from one of the counties of the State where he had been called the "human gorilla." He was dumb, and a low-grade imbecile, but had developed into a large, exceedingly powerful man. He was good-tempered and frequently helped in lifting the helpless imbeciles. He had not had fits, and he had not been noticed to be short of breath. There had been no indications of heart disease.

On Tuesday, November 6th, he had a fit and became cyanosed. He died in a few hours.

The brain was deeply congested, but in it there was nothing to account for sudden death.

The heart, which was shown to the Society (through the kindness of Drs. Kerlin and Wilmarth), is not much enlarged. The right chamber contained clots and the left ventricle was relaxed and empty. The valves were competent. The aortic segments were thickened below the copora-Arantii. The left ventricle from aortic to apex measured five inches. The walls were from three-eighths to one-half inch thick. The anterior portion of the left ventricle was extensively sclerotic. The extreme apex and lower portion of the septum show the same changes. There were small scattered patches of fibroid degeneration in other parts of both chambers. The condition of the coronary arteries was most extraordinary. The orifice of the anterior branch was narrowed to a pin-point by atheromatous swelling of the intima. For a quarter of an inch from the aorta the walls were greatly thickened, and the lumen almost obliterated. The first main branch was obliterated in its course. In the right auricular-ventricular groove the vessel was rigid and in places calcified and the lumen almost obliterated. The branch in the posterior-interventricular groove was represented by a small fibroid cord into the tube of which the smallest pin would not enter. The posterior coronary artery was quite pervious at its orifice. The branch which passed down the anterior interventricular groove was patent, but presented many flakes of atheroma. An inch and a half from the apex it was converted into a dense firm cord. The posterior division of this artery was completely occluded.

The arch was not dilated. There were a few flakes of fatty degeneration on the intima. The descending aorta was healthy. The kidneys were deeply congested, but not at all reduced in size. The arteries were not prominent; the renal arteries were not stiff.

Dr. Osler remarked that apart from the interest which pertained to disease of the coronary artery so extensive in a comparatively young man, there were other features upon which this case directly bore.

Instances of endarteritis, confined chiefly to the coronary vessels, are not very uncommon. These are usually regarded as syphilitic in origin, but in this instance this factor may be positively excluded. The question, too, of alcoholism cannot be suggested, for the man was under restraint all of his life.

Is it possible that a highly-developed muscular system could have been a factor in the production of the endarteritis, as we know it sometimes is in the scleroses of the aortic valves?

November 10, 1887.

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5. *Aneurism of thoracic aorta.*

Exhibited by Dr. M. H. Fussell.

The notes of this case, as a dispensary patient, were published in the *Medical and Surgical Reporter* of September 10, 1887.

The patient, Michael E., æt. 50 years, a carpenter. No history of syphilis. Presented himself in May, 1886, with a ready-made diagnosis of rheumatism of right shoulder. He complained only of pain in right shoulder, but on being questioned acknowledged dyspnœa on exertion.

On examination, visible pulsation of superficial arteries. Apex-beat of heart half inch outside nipple in sixth interspace; a very slight prominence was noted in region of right third rib and sternum, which plainly pulsated. There was dulness over this prominence and a very faint systolic whiff was heard. He was put on iodide of potassium fifteen grains, *t. d.* He did not return until July when he stated that he was perfectly free from pain and complained but little of dyspnœa.

He was then lost sight of until July, 1887. He said that he had been comparatively well until some time in January, 1887, when, after an especially hard day's work, he had severe pain, with a rather sudden enlargement of the tumor. The tumor continued to enlarge for about two weeks, when it appeared to the patient to be almost as large as then (July, 1887). He was suffering from extreme dyspnœa, severe distressing cough and pain.

A great prominence could be seen through his clothing at the upper part of his chest. The prominence extended from the clavicle on the right side downward to the third rib. It was dumbbell-shape, measured five inches in greatest diameter, three and a half inches over each enlarged portion, and three inches over the constricted portion. Pulsated markedly, the pulsation being expansile and isochronous with the systole of

the heart. No thrill felt nor bruit heard over tumor. A double murmur was heard over the region of the apex. Pulses differed markedly as shown by sphygmographic tracing taken at the time. He was again put on iodide of potassium and forced to rest (previously he had been going about). The iodide gave almost complete freedom from pain and patient seemed much more comfortable until about the beginning of October. He then grew rapidly worse, breathing labored; tumor enlarged one inch in each direction. He finally died of pulmonary œdema on October 26th.

Post-mortem, thirty-six hours after death. Chest only examined.

The tumor was depressed and flabby. On dissection the central portion of the tumor was found to be covered only by the skin and fibrous remains of the pectoralis major muscle. Over the whole tumor the muscle was much thinned.

On cutting through the left costal cartilages and loosening sternum at lower part, the entire upper median portion of the chest was found occupied by the tumor. The right lung was adherent to the tumor and to the pericardium. A few adhesions of left lung were also found fastening it to the sac. Both lungs were "waterlogged." Owing to limited time no attempt was made to dissect the tumor *in situ*, but it with all adherent parts was removed and dissected at leisure afterward.

The portion of the tumor external to the body was filled with rather soft clots. The remainder, and by far greater portion, contained no clots.

The external tumor presented the same shape as it did during life, a constricted portion lying in the situation of the first intercostal space, and two enlarged portions, the upper opposite the first rib and the lower opposite second and third ribs.

It measured six inches in its longest diameter. The upper portion measured three and a half inches, the lower four and a half inches.

The right half of the sternum was eroded from one inch below upper margin to opposite the third rib, together with at least one inch of the first and second ribs.

The sac sprung from the ascending portion of the arch of the aorta, one inch from the beginning, and involved the transverse portion as far as the left carotid artery.

Its greatest circumference, just below sternum, was thirteen inches.

The circumference where it joined the dilated aorta was nine inches.

Its length, or distance from aorta to sternum, was four inches at the lower margin. The cavity will contain two and a half ounces of absorbent cotton not very closely packed.

The aorta was greatly dilated, having a circumference of five inches at its origin. Its wall contained numerous patches of atheroma.

The heart was not greatly dilated, the greatest dilatation being in the right ventricle. The walls of the left ventricle were five-eighths of an inch thick.

Both the mitral and aortic valves were thickened. The tricuspid orifice admitted four fingers and a thumb. The superior cava was considerably constricted. No marked compression of either gullet or trachea.

In this specimen the vertebral artery is given off from the transverse portion of the arch of the aorta, between the left carotid and left subclavian arteries. *November* 10, 1887.

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6. *Aneurism of the ascending aorta.*

Exhibited by Dr. James Tyson.

This specimen was derived from an Irish domestic, æt. 76 years, who who died in the Philadelphia Hospital.

About the middle of March, 1888, while recovering from a cold, she was seized with pain in the left side. There was orthopnœa, cough, slight expectoration, and cardiac palpitation. These symptoms, probably due to the pleurisy of which such marked results were found at the autopsy, claimed little attention until a tumor appeared suddenly in the upper central præcordial region, a short time before admission, which was on March 27, 1888. A distinct impulse could be felt in the tumor, explaining the cardiac palpitations complained of, but no thrill.

On May 16th we noted enlargement of the superficial veins over the tumor, and a double murmur, most distinct at the aortic cartilage, and in all respects, except as to loudness, corresponding to the sounds heard at this location in health. There was percussion dulness to the right of the sternum and thence into the axilla and posteriorly throughout the right side. Resonance remained normal on the left side. Corresponding to the area of impaired resonance the breath sounds were much enfeebled. The pupils were unequal, the left being much the smaller. The patient complained occasionally of pain in deglutition, and of pain in various parts of the thorax, more especially in the right side.

On the next day she complained of a sense of constriction in the upper part of the thorax and a sense of suffocation. The dyspnœa continued to increase; on May 20th she was cyanosed about the face and numerous ecchymotic patches appeared upon the hands and arms. The breathing was jerking and the patient was comatose. This condition continued until death, which took place at 2 o'clock, of May 21st.

Autopsy.—The right thorax was almost completely filled with a straw-colored fluid, and the corresponding lung proportionately compressed.

Just above the pericardium appeared a tumor, three inches in diameter, which proved to be the aneurism involving the ascending limb of the aortic arch in the second position. The anterior wall of the aneurism was formed by a dense fibro-plastic mass, which substituted the aortic walls and the upper end of the sternum, which was so far eroded that it could not be raised without fracturing. The aneurismal sac was occupied by an exquisite laminated clot of great firmness.

The pericardium contained about an ounce and a half of turbid lemon-hued fluid, while in many places there was adhesion between the visceral and reflected surfaces. The heart itself was somewhat hypertrophied.

The most interesting feature connected with this case is, after all, not an unusual one, and that is the almost total absence of subjective symptoms until a very late date in the history, as there is little doubt that the pain and orthopnœa complained of on admission were due to the comparatively latent pleurisy and not to the aneurism.

November 10, 1887.

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7. *Aneurisms of the aorta. Two cases.*

Exhibited by Dr. G. E. de Schweinitz.

Case I. *Aneurism of the arch of the aorta; perforation into the œsophagus, together with two aneurisms of the abdominal aorta.*—James K., æt. 47 years, became an inmate of the out-wards of the Philadelphia Hospital, December, 1886. He remained in the out-wards until July of the following year, and was not submitted to medical examination, inasmuch as he never complained of any definite symptoms. During this month, after a fit of coughing, he was seized with hemorrhage from the mouth. He was conveyed to the medical wards, but died in fifteen minutes after admission.

Autopsy.—Body of a fairly well-nourished white male, with no unusual external appearances.

Thorax: Right pleural cavity obliterated by extensive old adhesions. Left cavity unchanged.

Right lung: Complete adhesions from apex to base. Several nodules as large as a hickory-nut and one cavity of the same size in the upper lobe.

Left lung contains a few nodules, partly cheesy and partly cretaceous, scattered through both lobes.

Heart and pericardium: Pericardium smooth. Left ventricle much thickened and hypertrophied. Right side of the heart and its valves unchanged. The mitral leaflets are thickened and contain a few patches of atheroma. The aortic valves thick and opaque; calcareous patches in the aorta immediately above them. The aorta directly above its commencement begins to be dilated and pouched, and just beyond the origin of the left subclavian there is a large aneurismal sac. This sac is attached to and has eroded the bodies of the adjacent vertebræ. The sac is partially filled with clots and has ruptured into the œsophagus. The opening is oval, one inch in its long diameter, and contains a plug of reddish clots. It is in immediate relation with the bifurcation of the trachea in front, the cartilage of which is in some degree absorbed.

Abdominal cavity: Liver presents no unusual characteristics.

Spleen small and the pulp tears readily.

Stomach contains one quart of partly fluid and partly clotted blood. No lesions in the intestinal canal.

Kidneys are both small. Capsules detach with difficulty; cortices narrow and pallid. Upon the surfaces are numerous yellow spots and the right kidney contains two small cysts.

Aorta: Immediately below the diaphragm the abdominal aorta is dilated, and on the left side there is a large aneurism situated along the sides of the vertebra, the bodies of which are extensively eroded. The aneurism passes downward, outward, and backward as far as the bifurcation of the vessel into the iliac arteries. The sac is completely filled with firm fibrous laminæ, closely packed one above the other. Immediately above the superior mesenteric artery, springing from the anterior face of the aorta, there is a pouch-like aneurismal sac, the size of a walnut. The aorta itself contains numerous atheromatous plates, as far down as its bifurcation into the common iliac arteries. Brain and cord not examined.

Case II. *Extensive mitral and aortic disease; atheromatous degeneration of the aorta; two aneurisms of the thoracic aorta.*—John O'N., æt. 51 years, admitted to the Philadelphia Hospital October 8, 1885.

History.—Father and mother died of rheumatism. Patient himself had had a number of attacks of rheumatism. He denied syphilis. He had been a soldier. He was admitted on account of the disease of the heart, and during his residence in the hospital, in addition to the physical signs of hypertrophy, with dilatation, four distinct cardiac murmurs could be differentiated, viz.: systolic and diastolic aortic murmurs and presystolic and systolic mitral murmurs. Toward the close of his life the heart's action became so feeble and irregular that it was no longer possible to separate these murmurs. A distressing symptom, for a long time present, was intense pain in the back, at the lower angles of the scapulæ. The patient died July 22, 1887.

Autopsy.—Body of a large, white male, which presents no unusual external appearances, except a curve of the dorsal vertebræ.

Thorax, lungs, and pleuræ: A few fibrous adhesions at the right apex; the pleural cavity contains a pint of blood-stained serum. Slight adhesions at the left apex, and the pleural cavity contains two quarts of similarly stained fluid. The right lung is crepitant throughout, the left is collapsed and pressed against the bodies of the vertebræ.

Heart and pericardium: The pericardium is smooth, and its cavity contains three ounces of blood-stained serum. *Heart* much enlarged, in a state of hypertrophy, with dilatation. Large, partially fresh and partially chicken-fat clots in both heart-chambers. Tricuspid valves smooth. The orifice admits three fingers and a thumb. Mitral orifice admits three fingers; the valves are shortened, thickened, and upon the attached margins are dense calcareous deposits. The aortic valves are shortened, and the leaflets at their attachments stiff with hard, calcareous material. Immediately above them the atheromatous degeneration of the aorta begins, and consists of a series of calcareous plates, closely joined together and extending far out into the innominate, carotids, and subclavians. The arch of the aorta is enormously pouched and dilated. The same conditions obtain in the descending aorta. Opposite the fifth dorsal vertebra a double aneurismal sac begins, which lies on either side of the vertebræ, extending down to the diaphragm. It is entirely filled with successive layers of densely packed fibrous clot. The bodies of the vertebræ are misshapen and eroded.

Abdominal cavity: Liver slightly enlarged, congested, and on section presents the appearance of nutmeg liver.

Kidneys: About natural in size, darkly congested, slight adherence of capsules.

Spleen: Normal in appearance.

The rest of the abdominal viscera, as far as noted, present no abnormal appearances. Brain and cord not examined.

I would briefly call attention to the following points of interest: 1. They are interesting and typical examples of what Dr. Sharkey would call multiple medical aneurisms. Three distinct aneurisms existed in the first case and two in the second. 2. The aneurism of the arch of the aorta, in the first case, apparently produced no marked symptoms until the perforation into the œsophagus and the hemorrhage took place. 3. The aneurism of the thoracic aorta, in the second case, and that of the abdominal aorta, in the first case, are typical examples of "cured aneurisms." *November* 10, 1887.

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8. *Mitral stenosis. Three attacks of chorea.*

Exhibited by Dr. W. Osler.

Frank N., æt. 13 years, a well-nourished, well-grown lad, presented himself at the Infirmary for Nervous Diseases in May, 1887, in answer to a post-card asking him to return and have his heart examined. He had been under treatment for chorea in 1884. The first attack was in the spring of 1881, and lasted several months. It was severe, as he could scarcely walk and lost the power of speech. The parents are positive there was no articular disease. He had a second slight attack in 1882, and a third in 1884, when he first came to the Infirmary, and at this time he was under observation for three months. The father and mother are healthy. A half-brother had rheumatism.

He had a mild attack of scarlet fever when he was two years old. The case, No. XXXIX., is briefly reported in my paper on the "Cardiac Relations of Chorea," in the *American Journal of the Medical Sciences* for October, 1887.

The condition at the time of examination in May was as follows: Face flushed; lips purplish on exertion. Can get about comfortably if he goes quietly. Is very short of breath if he runs. Feet have swollen. Præcordia is prominent, particularly at fourth, fifth, and sixth left costal cartilages. Wide area of impulse, not very forcible; apex in fifth, two inches outside nipple-line. Veins of neck full, and in recumbent

posture present a wavy pulsation. On palpation a marked thrill, maximum in fourth space to right of nipple, feeble in third, not to be felt in fifth. It distinctly precedes the impulse.

Percussion: Absolute dulness from lower of third above; and one and a half inches outside nipple-line, and to the right a full inch from the sternal margin.

Auscultation: A rough, grating, presystolic murmur of maximum intensity, in fourth space inside nipple; also heard below and outside the nipple. In whole apex region a loud, blowing systolic bruit heard as far as the posterior axillary fold. At the lower sternum the systolic murmur was also loud, probably transmitted. Second sound along the left margin of the sternum greatly accentuated. No murmur at the aortic cartilage; second sound feeble.

The lungs were clear. The liver was much enlarged, causing a prominence at the upper part of the abdomen. A very unfavorable prognosis was given.

Toward the end of the year dropsy set in; Dr. Hewish tapped the abdomen and punctured the distended legs. Death took place January 5th. I am indebted to the kindness of Dr. Hewish for the privilege of being present at the autopsy.

Face cyanosed; moderate anasarca, abdomen distended. Slight exudation into pleural sac and pericardium.

Heart very large, weighs sixteen ounces, transversely placed, resting upon the enlarged liver. The right chamber enormously distended; the auricle as big as the closed fist; the left ventricle not apparent anteriorly, but lies entirely behind the right chamber. The left auricle large, but not so distended as the right; the appendix is not seen without tilting up the heart. A solid clot, which filled the two hands, occupied the right auricle, and in the other chambers there were firm coagula. The cavæ very large—three fingers could be inserted into the inferior; the right auricle received a ball of eleven inches circumference; wall thick, and the trabeculæ of appendix much hypertrophied. The tricuspid orifice narrowed, admits the thumb to middle of the first joint. The segments have united at the edges; the chordæ look thin and stretched. There are no vegetations. The chamber of the right ventricle measures from the pulmonary ring to the apex four inches; the walls are hypertrophied, measuring one-half an inch. Pulmonary valves normal. Left auricle dilated, wall thick, three lines, endocardium very opaque. The mitral orifice is represented by a narrow slit at the bottom of a flat depression. The edges are serrated and rough with calcified nodules which extend into the anterior cusp, and to a less extent into

the posterior, making the entire curtain rigid and immobile. The left ventricle is small, measures two inches from aortic ring to apex. The aortic valves are small, and present a few warty vegetations at the corpora Arantii; aorta is smooth.

Lungs full in volume, brownish-red in color, very tough. At posterior part of left apex a firm hemorrhagic infarct, and at right apex a large one which has broken down and formed an irregular cavity.

Liver greatly enlarged, very firm, nodular on the surface, cuts with resistance; section shows a typical nutmeg appearance.

Kidneys enlarged, deeply cyanosed; very firm. Spleen large and firm.

November 10, 1887.

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9. *Aneurism of a branch of the pulmonary artery in a phthisical cavity.*

Exhibited by Dr. W. Osler.

The patient, a man, æt. 50 years, a miner, had been in the University Hospital for eighteen months with chronic phthisis, and subsequently went to the Philadelphia Hospital, where he died of hemorrhage from the lungs. There were signs of a large cavity at the left apex, with much induration. The antero-lateral portions of the lungs were emphysematous, and he suffered most from repeated bronchitic and asthmatic attacks. He had had several slight hemorrhages while under observation, and for several days before death there was constant bleeding.

The right lung contained a few tuberculous nodules, and was emphysematous. The left lung presented at the apex a cavity the size of an apple, with thick walls, much indurated and pigmented. The lining membrane was soft and in spots hemorrhagic.

At the apex of the lower lobe were two small cavities, which communicated with each other and contained blood-clot. Into the upper part of the larger cavity a pedunculated aneurism, the size of a large cherry, projected. It was surrounded with clots and the walls deeply stained and covered with fresh granulations; at the lower part there was a linear rupture three lines in length, through which water flowed freely when injected into the pulmonary artery. The greater part of the sac was filled with firm clot. The aneurism communicated with a branch of the pulmonary artery by a very small orifice.

Dr. Osler referred to the frequency with which aneurisms were found in phthisical cavities, and to their rupture as a very common cause of the late hemorrhage in consumption. *November* 10, 1887.

10. *Aneurism of the left middle cerebral artery.*

Exhibited by Dr. W. Osler.

During the last session Dr. Osler had read notes of twelve cases of aneurism of the large cerebral arteries which he had dissected, in eight of which fatal hemorrhage had occurred; in four the aneurism had produced no symptoms. The specimen presented came in the latter group. It was obtained from a man æt. about 60 years, who had been admitted from the out-wards of the Philadelphia Hospital with extreme debility and emaciation, and had only survived a day. An open cancer of the stomach, a large recent tubercular abscess in the right lung, and moderately extensive atheromatous changes in the vessels, were the principal lesions. In the left middle cerebral artery just before the main bifurcations an aneurism, the size of a pea, projected from the upper surface, and was imbedded in the gray matter just outside the anterior perforated space. It was sacculated, with very thin walls, and contained a small clot. A flake of atheroma existed on the convex surface. The vessels of the circle of Willis were not unusually atheromatous. *November* 10, 1887.

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11. *Large fibro-cystic goitre; hemorrhage; sudden death.*

Exhibited by Dr. W. Osler.

The specimen was removed from a woman, æt. 44 years, an inmate of the Philadelphia Hospital, who had had a large goitre for many years. Death occurred suddenly as she was walking about. The post-mortem record was as follows: Skin blanched; an immense goitre occupied the antero-lateral regions of the neck. Hemorrhage beneath the skin, particularly on the left side, where it also extended deeply between the cervical muscles. On removal of the sternum extensive extravasation continuous with that in the neck occupied the anterior mediastinum as low as the right auricle. Over the arch this formed a large solid clot, and from this it passed along the thoracic aorta, and along the trachea and œsophagus, particularly in the left side. The pneumogastric nerves were surrounded by clot. Above, the extravasation extended as high as the pharynx. The left lobe of the thyroid was soft and fluctuating, and, on section, presented large cysts filled with fresh blood-clot. The right

and middle lobes were firm and solid, cut with great resistance, and the stroma was in places calcified. In large areas the gland acini were visible, filled with solid colloid matter of a yellow-brown color.

November 10, 1887.

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12. *Combined mitral and aortic disease.*

Exhibited by Dr. W. Osler.

J. M., æt. 35 years, carpenter, admitted to the University Hospital, September 29th, with dyspnœa. When fourteen years of age he had a severe attack of rheumatism, which confined him to bed for five weeks. When nineteen he had a second attack. For three or four years past he has been troubled with palpitation of the heart on exertion. For four months he has been very short of breath; the feet were swollen occasionally, and he has been unable to work.

Physical examination showed great hypertrophy of the heart, the apex extending far into the sixth interspace. At the apex there was a loud systolic murmur, which increased in intensity toward the ensiform cartilage, and was well heard along the right costal margin. A loud echoing rumble filled up the diastole at the apex. At the aortic cartilage there was a feeble systolic murmur. In the second and third left interspaces, distinct double murmur, the diastolic portion transmitted as low as the sixth costal cartilage.

The case was regarded as one of mitral stenosis. The question of the involvement of the aortic valves was not definitely determined. The pulse was not collapsing, but was small and irregular. The diastolic murmur, though heard at the mid-sternum as high as the second cartilage, was always most intense in the third and fourth left interspaces.

During his stay in the hospital he had no dropsy; the dyspnœa improved. He was extremely nervous, and the slightest excitement would send up the pulse twenty or thirty beats. Vomiting set in early in November, and the patient died of gradual heart-failure.

The heart showed enormous dilatation, particularly in the right chambers, from which 34 ounces of fluid blood and clots were removed, including the amount which escaped from the great veins; 12 ounces of clots were removed from the left chamber; while in the chest the transverse measurement of the distended heart was 3½ inches. The organ showed enormous hypertrophy and dilatation. The right ventricle occupied the front heart, and the conus arteriosus reached into the third

and second left interspaces. The tricuspid orifice was moderately narrowed by the union and thickening of the three cusps. The pulmonary artery was not much dilated: it showed only a few flakes of fatty degeneration on the intima. The cavity of the left auricle admitted the closed fist. The appendix was occupied by a firm laminated thrombus. The mitral orifice, seen from the auricle, was a narrow, transverse slit one-third inch in length. The segments were united, greatly thickened, and calcified. On the ventricular side there was a curious anomaly. The anterior papillary muscle gave off only two or three small chordæ tendineæ, and the great mass of muscle was attached along the anterior mitral leaflet and at its base united directly with the muscular substance of the ventricular wall. The entire bunch of the chordæ tendineæ arose from a large and much-divided posterior papillary muscle. These chordæ were much shortened and thickened, and the narrowed mitral orifice was everywhere closely surrounded by them. The left ventricle was dilated and the walls hypertrophied. The aortic valves did not hold water. They were thickened at the edges, and partially united and foreshortened. The coronary arteries were normal. The weight of the heart without clots was 24 ounces.

The other organs showed no special changes.

Dr. Osler remarked that in addition to the general features of interest pertaining to this case, the anomaly of the anterior papillary muscle was worthy of special note, as we so rarely see deviations from the normal appearance in these structures. During the last session of the Society, Dr. Osler had shown a specimen identical in every respect, in which the anterior papillary muscle did not give off chordæ tendineæ, but was attached along the anterior mitral cusp, and at the base united with the muscular wall of the ventricle. *November* 10, 1887.

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13. *Cases of cardiac and renal disease.*

Exhibited by Dr. H. M. Fisher.

Case I. *Showing cardiac hypertrophy and parenchymatous nephritis.*—George H., æt. 39 years, colored, was admitted to the Episcopal Hospital in November, 1887. He had suffered from pain in the chest and dyspnœa on exertion for a long time. Only recently had there been much œdema of the legs and ascites.

His urine was highly albuminous, and there was a systolic apex and a diastolic basic murmur. Paracentesis abdominis was performed three or four times during his stay in the hospital, but the fluid rapidly re-accumulated, and, in spite of free purgation and diuretics, the dropsy, dyspnœa, and debility increased. The day before his death coarse friction was heard all over the præcordial region. Died December 17, 1887.

The *post-mortem* examination revealed rigidity and thickening of the semilunar cusps, marked atheroma of the aorta, and dilated hypertrophy of the left ventricle—the heart weighing 24 ounces. The pericardium was roughened and thickened, and adherent near the base of the heart. The kidneys were smooth on their surface, but showed marked diminution in their cortical substance. Their tissue was yellowish-red and much congested.

Case II. *Showing hypertrophied heart with mitral and aortic vegetations, and fatty and contracted kidneys.*—Eliza H., æt. 39 years, single, mill-hand, born in England, was admitted to the Episcopal Hospital in June, 1887.

Patient's ill-health appears to have dated from an acute rheumatic attack six months before her admission to the hospital. Upon her admission it was noted that she was pale and thin, and that there was slight œdema. Micturition scanty. Urine was pale, showed albumin in large amount, and epithelial tube-casts.

The second heart-sound was markedly accentuated. There were no signs of serous effusion into either pleural cavity when she was admitted, but later both pleural cavities were found to contain fluid, necessitating the tapping of the right chest, which was done by Dr. M. J. Lewis about four months after her admission. This operation was repeated twice at intervals of ten days or two weeks, and on each occasion alarming symptoms of prostration supervened. Later, general œdema, too, of the subcutaneous tissues occurred. During certainly the latter half of her illness in the hospital the quantity of urine passed was very small—during the last month before her death not averaging more than a pint daily, and for a day or two, perhaps a week, before her death, there was almost complete suppression of urine. Along with this diminished secretion of urine there were nausea and anorexia, but no violent headache or cerebral symptoms.

Diuretics of all kinds were tried, but utterly failed to promote increased action of the kidneys. Diaphoresis, on the other hand, was easily induced, but, prostration becoming more marked, efforts at caus-

ing free diaphoresis by means of the wet-pack, etc., had to be given up, as after each sweating patient was much exhausted.

Breathing became more and more oppressed, and for two or three days before her death her pulse was extremely thready and irregular. Died December 17th.

Post-mortem examination, twenty-eight hours after death.—General anasarca. On opening chest, about three pints of clear serum escaped, the right thoracic cavity being about two-thirds full of fluid, and the right lung being compressed to about one-third its natural size. Old pleuritic adhesions upon both sides of chest. Both lungs were congested and somewhat œdematous (the right lung markedly so), and showed emphysematous lobules. The lower lobe of the left lung showed a cicatrix (possibly pointing to an injury to the lung during aspiration), and near this point was an infarct of the size of a marble.

Heart: Pericardium roughened and somewhat thickened, and some flakes of recent lymph near the base of the heart. No pericardial adhesions. The left ventricle was pretty firmly contracted and its walls were much thickened. The mitral valve admitted the tips of two fingers with difficulty, and there were vegetations upon its surface.

The aortic valve did not appear to be entirely competent, was rigid and thickened, and showed vegetations. Atheromatous degeneration of the aorta above the valve.

Kidneys: Both kidneys weighed about five ounces, and were irregular in outline and yellowish-white in color. Capsule rather unduly adherent in both, and cortex diminished. Cicatricial depressions upon the surface of both, and rather marked congestion, particularly in the pyramidal portions.

Liver about normal in size, but in consistence unduly firm.

Spleen narrow, elongated, somewhat atrophic.

Dr. Meigs called attention to a paper in the *Lancet* of December 10, 1887, by Steven, in which the point is made that fibroid degeneration of the heart is, in the great majority of instances, caused by disease of the coronary arteries. Such a condition of the tricuspid opening as was found in Dr. Fisher's case is common enough, and cannot be considered as having been congenital, but was the result of dilatation. The safety-valve action of the tricuspid was fully described by T. Wilkinson King in a volume of the reports of one of the London hospitals many years ago. Sibsin, in his account of the anatomy of the heart (Reynold's *System of Medicine*), shows how much more liable the tricuspid is to be stretched than the mitral, both because the whole setting of the tricuspid opening is muscular while that of the mitral is one-third tendinous, and

because the muscle of the right heart is so much less thick than that of the left. If the tricuspid valve be found to leak very much, when a stream of water is projected against it, it can be made to hold almost perfectly by passing a string around the base of the opening and drawing upon it until the size is sufficiently reduced to be about normal, thus proving that the leakage took place merely because the opening had been enlarged by stretching. The article of Gull and Sutton is open to criticism in many particulars; for instance, the very name is a bad one—"Arterio-capillary Fibrosis"—for they say nothing of the state of the capillaries, except some brief mention of the Malpighian tufts being diseased. Dr. Johnson, too, showed in a later number of the *Medico-chirurgical Transactions* than that in which this article appeared, that the methods employed were faulty, and that the apparent fibroid condition of the external coat of the arteries which they describe was in truth due to the use of camphor and glycerin, in which their specimens were prepared. The article, however, has served a very useful purpose in calling general attention to the fact that in Bright's disease there are often very extensive changes of the arteries and arterioles. As illustrating the extensive disease of the arterial system and the variability of the clinical symptoms with which Bright's disease originates, he cited some cases.

I. A man of Indian descent, who had all the signs of Bright's disease—albumin and casts in the urine, enlargement of the heart, dropsy, orthopnœa, congestion of the lungs, etc.—and yet the most careful inquiry into his history would lead to no conclusion but that the first warning that he had had that his health was failing was that he became subject to colds, with cough, and this is not at all an uncommon mode of origin of the disease, it seeming sometimes to fall first upon the lungs.

II. A man died last summer in the Pennsylvania Hospital of the disease in a typical state of development, in whom there were found disease of the arteries of the brain, spinal cord, lungs, heart, liver, kidneys, spleen, and the larger arteries, including the radial, femoral, and aorta.

III. A patient died in the same hospital about a year ago with aortic regurgitation, and in whose urine neither albumin nor tube-casts were ever found. He declared he had been sick only a few months, and yet the post-mortem examination revealed a condition almost exactly parallel with that found in the second case mentioned—extensive disease of the arterioles in all the organs examined, and of the larger arteries, including a high degree of atheroma of the aorta throughout its entire length. Rheumatism was not the cause of the disease, and the post-

mortem conditions certainly pointed toward a close parallelism with the second one, the first departure from the normal having been probably the change in the arteries or arterioles in both instances. In the first case the clinical symptoms pointed first toward disease of the lungs; in the second, of the kidneys; and in the third, of the heart; and yet they might all be properly called Bright's disease. He wished to emphasize the statement that extensive disease of the intima of the vessels is found in these cases, if looked for, and that this would seem to point toward the condition being identical or intimately connected with the atheromatous condition so commonly found to exist in the larger arteries.

Dr. Meigs referred to the difficulty of diagnosis, and instanced a case in which it had been supposed that there was pericardial effusion, and the autopsy showed that the pericardial sac was obliterated, and the dulness upon percussion in the præcordial region was due to great thickening of the pericardium. In this case a surgeon had been called in consultation to discuss the propriety of tapping, which fortunately, however, was not attempted. *December* 22, 1887.

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14. *Two cases of ulcerative endocarditis.*

Exhibited by Dr. W. Osler.

Case I. *Extensive destruction of aortic segments; hæmoptysis; emaciation; and the physical aspects of advanced phthisis.*—A female, æt. 30 years. Was first seen in November last, complaining of shortness of breath, cough, and spitting of blood. She had been ill four weeks with these symptoms, and had lost flesh rapidly. When first seen she presented a typical appearance of advanced phthisis. Owing to her extremely debilitated condition no minute examination was possible, but auscultation of the lungs showed everywhere present numerous râles. Over the cardiac area a loud to-and-fro murmur of double aortic disease was plainly audible. The true condition was not suspected, but the case was thought to be one of phthisis, with heart disease. The temperature was not taken, but she did not seem to have much fever. The autopsy showed engorged but not diseased lungs. The heart weighed 13 ounces. It was greatly dilated, and all its chambers were filled with clots. The mitral orifice was a little narrowed, its edges thickened, and studded with a row of fresh vegetations. The aortic leaflets were extensively diseased, the left coronary leaflet almost destroyed, with

three large vegetations projecting from the remnant. There were no infarcts.

Case II. *Leg ulcers; ulcerative endocarditis of mitral valves; intermittent type of fever.*—A man, æt. 62 years, an ostler, was admitted to the hospital November 30th, for a leg ulcer. There was a history of a daily chill, followed by fever and sweating, since the 26th. On the evening of December 2d, when the temperature was first taken, it was 102°. On the 3d, at 8 A.M., it was 98.4°; at 3.30 P.M., in a chill, 105.4°; in the evening normal. On the 4th, at 7 A.M., 105°; at 3.30 P.M., normal. On the 5th there was no chill, and the temperature was normal throughout the day. On the 6th there was no fever till evening, when the temperature rose to 105°, and he had several chills. On the 7th he was heavy and dull, and could only with great difficulty be roused. The heart sounds were feeble, and in the third and fourth interspaces just to the left of the sternum there was faintly audible a rather indefinite systolic murmur. At 8 P.M. of this day there was a chill. He soon lapsed into complete unconsciousness, and died in this condition on the 9th. Throughout the time he was under observation the urine contained no albumin. At the autopsy the mitral valve was found extensively diseased, and covered with vegetations. There were no other lesions worthy of note. *January* 12, 1888.

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15. *Multiple aneurisms of aorta; external rupture.*

Exhibited by Dr. W. Osler.

On January 5th I lectured on I. H., æt. 62 years, who had been admitted to Dr. Janney's ward at the Philadelphia Hospital, about three weeks before, with a large tumor of the upper sternum. He had had syphilis when young, had served in the army, and had been a hard worker up to a month or so before admission. He complained of shortness of breath, dull pain in the chest, and attacks of suffocation at night. A large pulsating tumor, 8 by 9 inches, occupied the upper region of the chest and projected to a level with the thyroid cartilage. It had destroyed the manubrium, the costal cartilages of the first and second ribs, and the sterno-clavicular articulations. There was no thrill on palpation, but a diastolic shock was evident. There was dulness, which extended far to the right. No murmur. There was an old flesh wound (bullet) on the right pectoral, and from the bottom of this blood oozed. A small bleeding-point was also noticed at the lowermost part of the

tumor. The heart was not enlarged, sounds feeble, no murmur. The right radial could not be felt. The pupils were equal. From the 5th to he 10th he bled more or less constantly from the erosions, which gradually enlarged. On the night of the 9th he lost very much, and sank gradually, and died on the night of the 10th.

The heart was not enlarged, the walls of the left ventricle did not look thicker than normal. The aorta to the cœliac axis is dilated, and the intima very atheromatous. The arch presented a circular orifice a little larger than a silver dollar situated at and to the left of the orifice of the innominate, which led directly into the large sac. The left carotid given off just within the orifice. The aneurism lay upon the trachea, which was a little compressed, but it had grown anteriorly and destroyed the manubrium, inner third of the clavicles, and the cartilages of the first, second, and third ribs. The sac, which was the size of a cocoanut, was filled with clots, some old and laminated, but peripherally more recent. The right carotid and subclavian were given off from the sac. The pneumogastrics were not involved. Just beyond the left subclavian was a second aneurism, the size of an orange, filled with firm thrombi. The bodies of the third and fourth dorsal vertebræ were deeply eroded. Just above the cœliac axis was a third sac, conical in shape, projecting to the right, and in contact with the spleen. It, too, was filled with old and very dense laminated clots. *January* 12, 1888.

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16. *Anæmic necrosis of the heart muscle.*

Exhibited by Dr. W. Osler.

The specimen was removed from a man, æt. 23 years, who died of extensive pneumonia (four lobes affected) on the thirteenth day. The heart was not enlarged; valves normal; a few flakes of atheroma on the aorta just above the level of the sigmoid valves. Coronary arteries not atheromatous. Section of the septum showed an extensive area of anæmic necrosis, extending from the apex to the auriculo-ventricular ring, varying in width from two to three lines. It was nearer the right than the left ventricle. When fresh it had a dull gray-white appearance which contrasted strongly with the contiguous red-brown muscle. Close examination showed that the process was not uniform, but intervening strands of normal fibres separated larger areas of necrotic muscle. It was not specially soft; indeed, it cut less readily than the normal portions. On microscopical examination the fibres were found to be extensively changed,

the striæ and nuclei no longer visible, but coarse irregular granules occupied the entire substance. Stained with osmic acid, the anæmic areas remained colorless, while the contiguous fibres, slightly fatty, showed dark. There was fatty degeneration of many of the medium-sized arteries. *January* 26, 1888.

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17. *Multiple aneurisms of aorta.*

Exhibited by Dr. W. Osler.

Wm. S., æt. 50 years, laborer, was admitted to the Philadelphia Hospital in October, 1887, complaining of pain in the region of the heart and shortness of breath. Had had syphilis, and had served in the war. There was marked pulsation in the lower sternal region and epigastrium; no prominence. Dulness not much increased; note clear in manubrium. Sounds at apex clear and booming, soft systolic murmur with the first. In the fourth interspace within the nipple there was a soft double murmur, limited in area. The sounds at the aortic cartilage were clear. Pulse 116, small and irregular. The patient remained up and about for the greater part of the time, and complained only of the distressing tightness in the chest. The wide heaving impulse, with the feeble, irregular pulse, and without increase to any extent in the cardiac dulness, suggested a deeply-placed aneurism of the heart. The epigastric pulsation was so marked when he first came in that the question of aneurism of the descending aorta was suggested. A large aneurism of the thoracic aorta was not suspected. The physical signs remained unchanged except the double murmur at the fourth interspace, which was extremely variable and for a long time could not be heard.

The autopsy showed a medium-sized heart without valve lesion. Immediately behind it was an aneurism of the thoracic aorta the size of a cocoanut, over which the œsophagus curved. The aorta, slit open, presented five aneurisms, two small ones projecting from the anterior wall of the terminal part of the arch; a third, the size of an orange, at the first part of the thoracic portion had eroded the bodies of the third and fourth vertebræ; a fourth, the large one, was in contact with the five lower dorsal vertebræ, the ninth, tenth, and eleventh being extremely eroded. The fifth was a small sac, the size of a walnut, opposite the orifice of the cœliac axis. The arch was slightly dilated, and there were atheromatous patches along the entire length of the aorta.

February 9, 1888.

18. *Aneurisms of the splenic artery.*

Exhibited by Dr. W. Osler.

Sarah M., æt. 60 years, admitted January 26, 1888, in a condition of extreme debility, and died January 30, 1888.

At the autopsy there were no special lesions found; the heart was small; slight atheroma of aorta, and the kidneys were cirrhotic. On the splenic artery, about two inches from the axis, was an aneurism the size of a walnut, with calcified walls; on section this was found filled with stratified and calcareous thrombi. Three smaller ones, the size of peas, occupied the main divisions of the vessel in the hilus. They, too, were calcified. *February* 9, 1888.

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19. *Aortic valvulitis.*

Exhibited by Dr. F. A. Packard.

The specimen was obtained from the body of an Italian admitted into Dr. Hutchinson's ward at the Pennsylvania Hospital on account of double pneumonia. At post-mortem the specimen was found, the breath sounds being so noisy as to obscure the murmur (if one existed) during life. There is seen to be marked thickening of the aortic valve leaflets, with calcareous infiltration, both processes being plainly marked at their line of contact. No kidney or other diseased condition was found that could have produced aortic valvulitis; and the patient's occupation, that of a railroad laborer engaged in shovelling, was the only assignable cause for its production. *March* 8, 1888.

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20. *Cardiac degenerations and Bright's disease the result of changes in the intima of the arteries and veins.*

Exhibited by Dr. A. V. Meigs.

A man, æt. 54 years, was admitted to the Pennsylvania Hospital July 12, 1886, with the following history: He had always enjoyed good health except that about ten years before he had had an attack of rheumatism,

which, however, was so slight that he was never obliged to give up his work. He had used alcohol in moderation, and denied having had venereal disease. He was taken sick, about four or five months before admission to the hospital, with dyspnœa, cough with expectoration, and loss of flesh and strength, and some swelling of the ankles. There was a trace of albumin in the urine, but the microscopical examination was negative. The albumin soon disappeared, and was not again found during his life.

Physical examination gave the following results: Radial pulse aortic in character; there is forcible and heaving pulsation over a largely increased area in the præcordial region. The apex impulse is in the sixth interspace outside the nipple-line, and pulsation can be felt as high as in the second interspace, and slight pulsation in the epigastric region. At the apex the sounds are booming, and there is a loud, harsh murmur synchronous in time with the aortic thrust. At the aortic area there is a double murmur, neither portion of which is very loud. The systolic murmur becomes harsh at the upper part of the sternum, and both murmurs are audible in the carotids and subclavians, the systolic being much the louder. There is visible arterial pulsation in the neck and of the radials. The jugular veins are swollen, but not unusually so, and there is no venous pulsation. Lungs filled with sibilant râles, and there is some dulness at areas both anteriorly and posteriorly. The liver dulness natural, and the splenic slightly increased.

The following inferences in regard to the case were recorded at the time: Disease is probably vascular in origin there is aortic obstruction and regurgitation and mitral regurgitation. The heart is enlarged, the enlargement preponderating upon the left side, and the vessels atheromatous. Lungs congested and bronchitis exists. He died October 4, 1886.

Post-mortem: Lungs somewhat adherent to chest wall and emphysematous and congested posteriorly. Heart much hypertrophied and distended with soft dark clots. The aortic valves thick and puckered, and the orifice narrowed. Mitral flaps not markedly diseased. Right side showed no valve changes. The papillary muscles in the left ventricle showed, upon section, marked fibroid degeneration, and there were spots, one in particular, in the anterior wall of the ventricle, which showed what seemed to be a less degree of the same degeneration. No such appearances could be detected in either the papillary muscles or walls of the right ventricle. The papillary muscles of the left ventricle, when viewed in cross section, exhibited numerous holes of a size, and looking as if they had been produced by a pin being stuck in the tissue.

The aorta was much thickened, though not pouchy, and its ascending portion contained some calcareous plates. The arch and descending portion, and the abdominal aorta and all its branches, including the cœliac axis, mesenterics, renals, primitive iliacs, and iliacs after their division, as well as many smaller vessels, were markedly thick and uneven upon the inner surface. The abdominal aorta on section showed at least one minute cavity, probably an atheromatous abscess. The internal mammary and coronary arteries were very markedly atheromatous. Examination of the veins showed that the inferior cava was much thinner at some positions than others, though everywhere the lining of the vessel was smooth. One of the primitive iliac veins, though it looked, so long as unopened, natural, exhibited, when slit open, two bands which divided it into three separate channels, and must have reduced its carrying capacity by at least two-thirds. Another vein of about the size of a goose quill was found, which was four or five times as thick upon one side as the other, the thick side being like a rigid rod, and the other soft and flaccid as veins usually are. Stomach seemed natural. The liver granular and congested, and the spleen dark, congested, and firm. The kidneys congested and slightly cystic.

Sections were prepared for microscopical examination from the kidneys and their arteries, both large and small branches, from the lung, liver, heart, abdominal aorta, inferior vena cava, and a branch of the right renal vein. The sections from the heart included cuts, both transverse and longitudinal, of the posterior papillary muscle of the left ventricle, one including the anterior flap of the mitral and the left coronary flap of the aortic valve; one from the anterior wall of the right ventricle, including an artery; one about one-third or half way down the interventricular septum, including the wall of the left ventricle and arteries; and a separate section of the first descending branch of the left coronary artery. These sections all showed ordinary degenerative changes, such as would be expected, the kidney exhibiting in a marked degree parenchymatous change, and containing numerous casts, both in the cortical and medullary tubules. Some of the branches of the coronary vein distinctly exhibited thickening of the lining coat, and in one in particular, from the anterior surface of the left ventricle, there were spots which were so much thicker than the intima at most parts of the circle as to be very striking. These were made up of whorls of tissue containing nuclei, and in many respects resembled the so-called nests of epidermoidal cells. The point, however, to which it is particularly desired to call attention, is that all the various sections showed to a greater or less extent a peculiar morbid condition of the tunica intima

of the arteries and arterioles. This consisted in an irregular thickening, often, in small arterioles, to be plainly distinguished as lying entirely within the plicated membrane, and in larger arteries recognizable by the naked eye as belonging to the lining of the vessel, and containing in many instances a greater or less number of nuclei imbedded in a structureless matrix.

The sections from the papillary muscle showed in marked degree the appearances of fibroid degeneration, there being great increase of the fibrous material, this ramifying from centres in various directions amongst the muscle bands, and in spots total disappearance of the muscular tissue had taken place. In others, again, the sheaths of the fibres were still distinct, but their interior was filled with a material evidently different from that constituting the healthy bands often visible in the same field. In some cases these muscle sheaths, when seen in cross section in particular, seemed to have within them vacuoles, or the contained material was transparent. The cross markings upon the muscle bands were disposed to be indistinct, and many fibres seemed narrow and some pointed toward their extremities. There was marked brown atrophy, the pigment being visible at the poles of the nuclei both in longitudinal and transverse section. The muscle nuclei appeared in many instances to be much swollen. No large sized arterioles were visible, but in the transverse sections in particular there were a great many large roundish spaces which were surrounded by fibrous tissue containing nuclei. Though their walls were very thin, these were probably veins, especially as quite a number of them were filled with blood corpuscles. The endocardial layer varied much in thickness, and the outer portions in particular were in places infiltrated with round cells, and at spots where the increased thickness was great the muscular fibres underneath were much degenerated, in some places the sheaths seeming to contain only granular matter and again presenting the already mentioned apparent vacuoles.

It is very interesting to endeavor to discover, among the complexity and apparent confusion of organic changes found in this man's body, what was the primary disease, and certainly the attainment of success in such a search would be the first step in the direction of our being more happy in our therapeutic management of such cases than we now are. He had had bronchitis, emphysema, and catarrhal pneumonia, for there was distinct catarrhal infiltration of the pulmonary alveoli; valvular disease of the heart and fibroid degeneration; fatty infiltration and atheroma, with calcareous degeneration of some of the coronary vessels. There was also interstitial hepatitis, parenchymatous and cystic disease

of the kidneys, and inflammatory bands obstructing one of the largest (the primitive iliac) veins in the body, with further evidence of a diseased condition of the venous system (thickening of a branch from the inferior vena cava and thickening of the intima of some of the coronary veins of the heart). Lastly there was the disease of the arteries, consisting of the changes described in the intima, and found in all the organs and tissues in which they were sought for.

The conditions might be said by some pathologists to be due to syphilis, as there was outgrowth of the lining of the arteries containing cellular elements, but this seems a mere begging of the question, as the man denied ever having had the disease, and it is now acknowledged by some of the best authorities that such outgrowth is often due to other causes. A few years ago the case would have been said to be one of valvular disease of the heart of rheumatic origin, and all the other lesions to be secondary to this cause; but this seems a very weak explanation, as the patient never had any rheumatism, except some slight vague joint pains which lasted only a few days and never prevented his working as a day laborer. This hypothesis, therefore, would seem very far-fetched, and it is much easier to believe that the valvular disease was but part and parcel with the atheromatous changes found so extensively in the arteries, for what are the valves but reduplications of the lining portions of the arteries and heart?

Dr. Johnson, and those who accept his views, would probably say the case was one of parenchymatous disease of the kidneys, and that all the other pathological lesions were secondary. He says (*Medical Lectures and Essays*, by George Johnson, London, 1887, p. 653), "During the progress of chronic Bright's disease, the bloodvessels in the kidney and in many other tissues and organs undergo very interesting changes, but these occur later, and less constantly, than those which affect the secreting tissues of the gland," and again (p. 700) "the primary and essential structural changes consist in a desquamation, disintegration, and removal of the renal gland cells." In this case, however, there were no symptoms developed during life to draw attention to any disease of the kidneys, except that for a brief time after his admission to the hospital there was a trace of albumin, without casts, in the urine; the absence of any of the ordinarily recognized clinical symptoms of renal disease was a marked feature in the life history of the patient, the pathological lesions discovered after death, and suspected and recognized during life in other organs, were so much greater in extent and so overshadowed everything that might be attributed to the kidneys, that it seems very

unwarranted to assume that the kidney lesion, which was so latent, could have been the point of origin of the pathological train.

What, then, is left as an explanation of it all? The state of the blood-vessels which was found so widely diffused. There was evidence of marked disease of the veins as well as the arteries, and then the lesion, of all others, which was most extensively diffused, and which could be most easily demonstrated, was the change of the tunica intima of the latter. When several experts examine a section of an artery there will often be much difference of opinion expressed in regard to the state of the muscular coat, whether it is hypertrophied or not; and if hypertrophied, as to how much; but if the intima of an artery is seen to be twice as thick upon one side as on the other, every one must agree that the condition is a morbid one, or the section has been cut obliquely. This change of the intima has not been commonly noticed, and when it has been it has not been given by any means the prominence due it. Johnson, even in his later utterences, does not correctly represent it, for he says (*Medical Lectures*, etc., loc. cit., page 695):

"The thickening of the arterial coats is due to an excess of the normal tissue, with no appearance of structural change or degeneration. The increase of the inner longitudinal layer of the hypertrophied renal arterioles is due to a thickening of the tunica intima. The intima is composed of three distinct layers—an outer wavy elastic layer, in contact with the muscular coat; an inner endothelial layer; and an intermediate layer of delicate connective tissue . . . the thickening of the intima in cases of granular kidney is caused by an increase of the connective tissue between the elastic and endothelial layer. . . . It is always associated with hypertrophy of the muscular coat, and bears a constant relation to it. The structural characters of the thickened intima are remarkably uniform, its parallel fibres passing in the direction of the axis of the artery, and at right angles to the circular muscular fibres, while the canal of the artery retains its uniform and natural diameter throughout its entire course."

Again, at page 698, he says: "On comparing the renal arterioles with those from other tissues this remarkable difference is apparent, namely, that while the hypertrophy of the muscular walls occurs alike in the arterioles in all the tissues, the thickening of the intima is found, so far as I know, only in those of the kidney."

No one has done more for the advancement of renal pathology than Dr. Johnson, and yet in the passages quoted he makes a number of statements which can by no means be substantiated. In the present state of knowledge no one has a right to assume that the thickening of

the intima is due only to an excess of normal tissue, and is merely an increase of the connective tissue between the elastic and endothelial layers, for in elderly people it is almost always impossible to distinguish any endothelial layer as a positive boundary; and frequently, when the process has reached the further stages of its development, in the arteries of the heart for instance, there is extensive degeneration of the walls of the vessels, this extending deeply into the muscular layer. The existence, too, of hypertrophy of the muscular coat of the renal arterioles cannot always be positively demonstrated when there is evident increased thickness of the intima. The most marked characteristic of the thickening of the intima is its irregularity, its giving rise to various hills and hollows upon the inner surface of minute arterioles, just as can be seen in any marked instance of atheroma of the aorta or other large vessel. It is not worth while to repeat, after what has already been said, that the thickening of the intima is very far from being confined to the renal arteries.

The case as detailed would have been considered, without doubt, as one of Bright's disease with enlarged kidney, if the real lesions alone had heen examined; of valvular disease of the heart, due to endocarditis, if the gross appearances of that organ alone had been considered; and of fibroid degeneration, if the sections of the papillary muscle alone had been examined.

There is a communication in a late number of the *Lancet* (Lectures on Fibroid Degeneration, etc., of the Heart, and their Association with Disease of the Coronary Arteries, by J. L. Steven, December 10, 1887, and subsequent numbers), in which the author endeavors to show by the citation of cases that fibroid degeneration is, in the great majority of instances, due to disease of the coronary arteries. Why, then, is it not much easier and more natural to suppose that lesions such as those described are due to the condition of the arteries which is found to exist to so great a degree in so many different organs, and that the attack which has its origin in them becomes a case of Bright's disease, one of emphysema of the lungs, of valvular disease of the heart, or fibroid degeneration, or some other degenerative disorder, according as the arterial change takes possession of one or another of the organs?

It would seem, too, upon general principles, that it is much more likely that the intima is earlier attacked than the muscular layer, and suffers more, and causes more serious disorder, for the intima is supposed to constitute the walls of the capillaries, which have no muscular coat, and this would, perhaps, go to explain entirely, or in part, the fact of the

destruction of nutrition, which takes place in the organs most seriously affected.

The end to which these observations tend is to show that often Bright's disease, and some forms of valvular heart disease, besides many other chronic degenerative processes, are not separate entities, as has been supposed, but merely the varying expressions of a disease of the arterial system which probably has its origin in the tunica intima. What the cause is which sets this condition in motion must be, in the present state of knowledge, a matter of speculation. *March* 8, 1888.

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21. *Atheroma and atheromatous ulcers of the aorta, with aneurisms. Two specimens.*

Exhibited by Dr. G. E. DE SCHWEINITZ.

CASE I.—Mary S., æt. 80 years, was admitted to the nervous wards of the Philadelphia Hospital November 9, 1887, and died about three weeks later. At the time of admission she was the subject of a right hemiplegia of some standing. Her family and personal history was not obtained. The cause of death appeared to be gradual exhaustion.

At the post-mortem the following conditions were observed:

Brain: Section through the cerebrum revealed an old hemorrhagic cyst, involving the anterior portion of the optic thalamus and the adjacent portions of the internal capsule and lenticular body. The dura mater was not adherent. The ventricles were dilated with serum. All the vessels of the brain were atheromatous.

Lungs: Crepitant throughout.

Heart: Aortic valves atheromatous. On the mitral valve spots of atheroma, and the edges curled. The aorta was everywhere atheromatous, and opposite the tenth dorsal vertebra there was a small aneurismal pouch filled with reddish clot. The rest of the organs, except the kidneys, in which the lesions of an initial nephritis were manifest, as far as examined, showed no gross changes.

CASE II.—B. B. D., male, æt. 54 years. Was admitted to the Insane Department of the Philadelphia Hospital, December 11, 1886, and died November 19, 1887. His father died of phthisis, his mother of pneumonia. He had brothers and sisters living, who were mentally and physically sound. His occupation had been at various times that of travelling agent, railroad conductor, and carpenter. In May of 1886 he

began to have epileptiform convulsions. These convulsions were marked by the absence of any aura or cry, the spasm was tonic, the cyanosis of the face extreme, and there was but little frothing at the mouth. During his hospital life his mental state was that of marked dementia, and his habits were most filthy. Each convulsive seizure was followed by great physical prostration, which deepened gradually until death from exhaustion took place.

Post-mortem, twenty-four hours after death.—No unusual external appearances.

Lungs: Emphysematous; pleural cavities unchanged.

Heart: Pericardium smooth. Left ventricle firmly contracted, and hypertrophied. Blood in heart-chambers fluid. Right side and valves normal. On mitral valve patches of atheroma, and the aortic leaflets stiff with calcareous deposit, while above the valves the vessel was extensively atheromatous. Both coronary arteries highly calcareous.

Abdomen—Liver: Tunic congested; capsule smooth. No gall-stones. No lesions in the stomach and small intestines. The mucous membrane of the large intestine was thickened, congested, and showed numerous spots of ecchymosis.

Kidneys: Interstitial nephritis, shrunken cortex, granular surfaces, adherent capsules, and numerous minute cysts.

Spleen: Large, pulpy, soft.

In the thoracic aorta there were numerous patches of atheroma and atheromatous ulcers. In the abdominal aorta, just above the cœliac axis, this condition was most marked. Upon the right side of the aorta, below the cœliac axis, the active disease of the two inner coats of the vessels has allowed the distention of the outer coat into a small aneurismal sac, filled with reddish clots. The diseased condition of the vessels extends far down into the iliacs.

Brain: Bilateral external pachymeningitis, with extensive adhesion of the dura on each side of the longitudinal fissure. Quite general subarachnoid effusion, amounting to more than a pint. Over the convexity of the brain a thick milky-white opacity of the pia mater and general distention of the veins. The circle of Willis was universally atheromatous. The lateral ventricles were distended, and also the veins of Galen, and the ependymal veins. One spot of softening in the right optic thalamus at its posterior part. No other gross lesions discovered.

These specimens show nicely the development of an aneurism from preëxistent disease of the arterial coats. The sacculations have occurred when the active disease has destroyed the intima and media, and thus allowed the distention of the outer coat. *April* 12, 1888.

22. *Aneurism of the arch of the aorta bursting into the primary bronchus.*

Exhibited by Dr. A. Edwards.

John H. was admitted to my wards in St. Joseph's Hospital January 4, 1888, and died January 27, 1888. Upon admission the left pleural cavity was filled with serum. Three pints were removed by paracentesis, producing marked relief but not reducing the dyspnœa.

A murmur was now audible at the aortic cartilage, with a wide area of distribution, a thrill, bruit, and expansile pulsation were present—in fact, the ordinary physical signs of aneurism were well marked.

Melæna arose a few days before death, which occurred suddenly, preceded by a violent fit of coughing, sudden gush of blood from the mouth, and death in ten minutes.

Post-mortem, twelve hours after death, by the Resident, Dr. Otto. Heart and lungs alone examined.

Lungs: Right congested; pleural nominal. *Left* pleural sac filled with fluid, lung bound down to the upper posterior surface of the pleural cavity by firm adhesion; many adhesive bands spanned the pleural cavity.

Heart: Slight pericardial effusion; heart in a condition of dilated hypertrophy; incompetent tricuspid and mitral valves. Aortic and pulmonary artery valves competent. From the ascending and transverse aortic arch sprang a large aneurismal sac, which had ulcerated into the primary bronchus, thus causing sudden death. The cardiac valves, while they were rendered incompetent by the dilatation of the cavities, practically were still histologically unaltered, the specimen in this particular following the general rule of presenting normal valves with aneurism of the arch. *May* 24, 1888.

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23. *Tricuspid malignant endocarditis.*

Exhibited by Dr. Kirby.

Mrs. S., æt. 24 years, was admitted to the hospital October 29, 1887, with the following history: Married at nineteen; perfectly well previously. Had six miscarriages, which I afterward learned were the

result of criminal abortions. In June of 1886 had a miscarriage followed by peritonitis. Ever since this has had chills and fever, and severe night-sweats.

On admission patient was suffering from severe dyspnœa, sweating, hurried pulse, rapid and labored respiration. Apex-beat displaced to left of nipple; distinct thrill. Blowing presystolic murmur heard over base and apex. Presystolic also heard loudly at xiphoid. Faint systolic murmur heard at the aortic cartilage. Urine contained hyaline and granular casts.

Patient died one and a half months after admission. During this time the marked chills, great sweats, and dyspnœa were the most prominent symptoms.

Post-mortem: Pericardium contained eight ounces of clear serum. Heart normal size. Chambers contained post-mortem coagula. Right auricle large, endocardium smooth. Tricuspid orifice wide. From above a large, irregular vegetation projects from the anterior leaflet of the tricuspid valve. On the anterior segment there is a large, irregular vegetation occupying fully half of the valve and projecting from the edge in large, warty excrescences. These are yellowish-gray in color and distinctly gritty. At the edge of the vegetation is a soft hemispherical swelling, the size of a small pea, found on examination to contain pus. Attached to the anterior papillary muscle are two broken chordæ tendineæ, the ends of which are covered with vegetations. On the posterior segment there is a small eroded patch, covered with grayish vegetations, size 2 by 3 lines. All the other valves normal.

This case is particularly interesting:

1. In the situation of the lesion.

2. In the possibility of having had its origin in a pyosalpinx, the condition of the tubes being probably the result of miscarriage.

June 14, 1888.

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24. *Aneurism of aorta—two specimens.*

Exhibited by Dr. W. Osler.

A. B., æt. 56 years, had been an inmate of the Philadelphia Hospital out-wards for several months. He had had repeated attacks of dyspnœa, associated with lividity and signs of cardiac dilatation. In one of these attacks Dr. Tulley, resident physician, took twenty-five ounces of blood from his arm with great relief. He died of hemorrhage. The autopsy

showed a large aneurismal dilatation of the entire arch. The aortic valves were stiff, but not shrunken. The heart was not much hypertrophied. The posterior wall of the aneurismal sac was formed in part by the trachea and bronchi, and the cartilaginous rings were plainly exposed on the inner surface. Between the first and second rings of the left bronchus a perforation had occurred, through which the hemorrhage had taken place. The second bronchial ring had projected with a rough, sharp edge, and had either torn or worn through the other side of the bronchus into the œsophagus, in which there was a perforation the size of a five-cent piece. Pressure had doubtless much to do with this. The orifices of the vessels were free.

The second specimen was from a man æt. 56 years, who died on the same day as the preceding one, and who had also been an inmate of the hospital for many months, suffering from occasional attacks of dyspnœa. In one of these attacks he was given a hypodermic injection, immediately after which he fell over dead. The aorta was slightly dilated, and at the upper portion there was an orifice of the size of a half-dollar piece, which led into a sac as large as an orange. This lay beneath the sternum, which was eroded, but not pushed forward. In more than three-fourths of its extent the sac was obliterated by firm laminæ of fibrin. The innominate and left carotid arteries were given off just within the orifice of the sac, and the left subclavian, which left the aorta close to the mouth of the sac, was obliterated for a half-inch of its course. The heart was not hypertrophied, and the valves were normal.

October 11, 1888.

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25. *Hypertrophy of the heart.*

Exhibited by Dr. J. H. Musser.

In this specimen we have a perfect example of the condition of the heart.

The patient, æt. 57 years, a barber, white, was admitted into my wards with pronounced uræmia and a moderate degree of anasarca. Some months previously he had been in the hospital with bronchitis, but has otherwise been very well. He has been a very hard drinker and has had gonorrhœa, but denies syphilis. A short time after admission an apparently causeless vomiting began, and with it occasional delirium was noted. The anasarca was only of one week's duration. With the delirium dyspnœa developed, but in moderate form only. Eight days after

admission he had an epileptiform convulsion. This recurred, dyspnœa became more and more marked, his mental condition never satisfactorily improved, delirium always being present. On the 9th he died, after a day of most extreme uræmic asthma, from sheer exhaustion. The urine was scanty, sp. gr. 1006, contained a considerable amount of albumin and hyaline casts. The heart, as you observe, weighs eighteen ounces, and shows enlargement in all its walls, and particularly of the left ventricle, which averages seven-eighths of an inch in thickness. There is some slight atheroma of the aorta, but no disease of the coronary arteries can be detected. In addition to the hypertrophy of the heart, well-marked granular contracted kidney was found.

The case is of interest in connection with the paper that Dr. Loomis recently published. He believes that most patients with Bright's disease die of a dilated heart. While in all probability many cases do, such a condition of the heart muscle is undoubtedly dependent upon the condition of the coronary arteries, independently of the kidney disease. If the coronary arteries are free from disease, and the nutrition of the patient is preserved, the heart will be hypertrophied, in contracted kidney. It is not so much the lesion of the kidney as it is the condition of the coronary artery that determines the state of the heart.

In other words, the point I wish to make is, as previously laid down by Johnson and Gull and a host of others, that the heart is hypertrophied in cases of granular contracted kidney. *May* 23, 1889.

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26. *Typhoid fever; old and recent endocarditis; cirrhosis of the liver in a lad.*

Exhibited by Dr. J. H. Musser.

The specimens presented from this case are of peculiar personal interest to myself, for during the past eight years the lad has been under my own personal observation, and for a considerable time under the observation of my friend, Dr. Hughes. As early as 1880, when seven years old, he was brought to the University Hospital Dispensary, on account of mitral disease, with muscle failure. One year after that I treated him at his home, through a furious attack of pericarditis, during which I almost despaired of his life on account of the large effusion. He recovered from this, and once I myself, and once Dr. Hughes, attended him since then through attacks of pneumonia. We both recognized

the fact from the first that the cardiac lesion was mitral, causing both obstruction and regurgitation. Our notes indicate this, and that the murmur occupied the usual positions, and had the usual characterteristics of these lesions. At no time, however, was a thrill noted.

The little fellow lived with his aunts, and on account of his weak condition was fed on milk punches during a considerable period of his youth, particularly from the sixth to the twelfth year, while I know personally he partook frequently of the beer which the family were accustomed to have on hand. I had not seen him for at least three years, when, on the 11th of October, this year, he was admitted to my wards in the Presbyterian Hospital.

Note on admission: He is now fifteen years of age, and has been working for some time in a factory. Two weeks prior to admission he began to feel sick, but he did not give up until five days ago. He had no diarrhœa, however, nor epistaxis, nor abdominal pain. He was admitted five days after the fever began, and his evening temperature was $104\frac{2}{5}°$. The splenic dulness extends from the seventh to the ninth rib. The liver is not enlarged. Abdomen slightly tympanitic. His mind is clear, his face flushed, marked pulsation of the vessels of the neck is seen, and a double mitral murmur readily elicited. Respiration 32, pulse 120, fairly strong.

October 12. Morning temperature 104°, evening 100.3°; intellect good, abdomen more tympanitic; pulse 100, respiration 30, in the morning; 118 and 38, respectively, in the evening. Bowels moved three times, watery, yellowish, offensive stools. Until the 16th no marked change took place, temperature remaining high, ranging from 102° to 103°.

16*th.* Slept well, mind clear. Spots on abdomen since 14th. Cardiac impulse in fourth and fifth interspaces, apex beat half inch outside nipple line. Loud systolic murmur transmitted to axilla. Pulmonary second accentuated. Veins over lower thorax and abdomen much enlarged.

On the morning of the 19th temperature fell to 99.3°, and from that until the 23d ranged from 100° to 101°, morning and evening, respectively. He appeared to be better, his mind was clear, and he had no diarrhœa. He slept well. On the 20th a new crop of spots appeared, and on the 21st it was noted that the spleen could be felt readily at the edge of the ribs. Some cough began this morning. On the 23d fever became intermitting, and ranged as follows: 23d, morning 100°, evening 103°. 24th, A.M. 101°, P.M. 104°. 25th, A.M. 98.2°, P.M. 106°. 26th, A.M. 97°, P.M. 103.4°. 27th, A.M. 102°, P.M. 98°. Death took place on that day. During these days the pulse rate varied

slightly with the temperature, on the 23d and 24th being 100 in the mornings, and 126 to 138 in the evening. On the 25th, A.M. 96, P.M. 140. 26th, A.M. 112, P.M. 168. 27th, A.M. 160, P.M. 180. Respirations about 36 to the 26th, then they ranged up to 50 and 60 at different times. The general condition remained about the same during the first ten days. When the paroxysmal fever was observed the pulse became feeble, and the impulse weak. Delirium became marked, and more or less collapse, with the morning reduction of the temperature, ensued, it being most pronounced on the morning of his death, and from which he did not rally.

The urine had been examined repeatedly throughout the attack, and at no time were casts found, and only albumin in amounts that could be accounted for by the condition of the patient.

Autopsy, by Dr. Griffith, ten hours after death. Emaciated, abdomen distended, discolored by post-mortem change. On section, intestines and stomach distended by gas. Peritoneum normal. Diaphragm attached to the fifth rib on the right side, and the sixth on the left.

Thorax: The left pleural cavity contained some clear, yellow serous fluid. The left lung was slightly adherent to the chest-wall by recent lymph. The anterior surface of the upper lobe was covered with fresh hemorrhagic lymph. The right lung had a few fresh adhesions, and contained a few ounces of fluid in the pleural cavity. On section of the pericardium old lesions of pericarditis were found over the anterior surface of the left ventricle, covering an area of about one inch. The parietal and visceral layers were intimately adherent. The heart was enlarged, and the right dilated and full of fluid.

Lungs: The left upper lobe was entirely solid, and on section cut like leather; lower lobe crepitant, except a small portion of its upper aspect. The right lung was crepitant and congested. Some spots of pneumonic congestion were seen, which did not crepitate, at the junction of the upper and middle lobes.

Spleen was enlarged, soft, full of blood, containing several fresh infarcts; measured 5½ by 2½ inches, and weighed 7½ ounces.

Liver weighed 2 pounds 13 ounces. Surface nodulated along the edges, presenting the well-known appearance of hob-nail liver. On section cut with a gritty feel; the overgrowth of connective tissue, characteristic of cirrhosis, was well marked. The acini were distinct, full, and fatty. The overgrowth of connective tissue defined very well areas of hepatic tissue.

Kidneys normal, capsule slightly thickened, but not adherent. Gall-bladder distended, ducts normal. Mesenteric glands enlarged. The

cæcum contained some ulcers, and was congested slightly ; its glands were enlarged. Peyer's patches were swollen and ulcerated. Beyond the cæcum, in the first two feet of the ileum, there were a few congested solitary glands, but not markedly infiltrated. Peyer's patches in the jejunum were quite distinct, slightly red, presenting a shaven-beard appearance.

Heart: The right auricle was full of clotted blood; the right ventricle contained chicken-fat clots. The auriculo-ventricular opening on the right side admitted two fingers readily. The mitral orifice was contracted, scarcely admitting the little finger. The leaflets were thickened, the cordæ tendineæ were contracted; along the insertion of the cords numerous fresh granulations were found, which were soft. There was no loss of tissue by ulceration. The remaining valves were normal. The heart muscle was healthy.

Brain and *spinal cord* not examined.

Summary.—Old pericarditis, old mitral disease, cirrhosis of the liver. Recent endocarditis, pleuro-pneumonia, and lesions of typhoid fever. Recent infarcts of the spleen.

Several matters of interest are worthy of being called to your attention. First, the occurence of cirrhosis of the liver, of alcoholic origin, in a boy of fourteen, which had developed without symptoms thus far, and with the only sign of enlargement of the abdominal veins. Second, the intermitting fever possibly marked the onset of pleuro-pneumonia and endocarditis, which, from the appearance of the lesions, were quite recent. Third, the proof at the autopsy of such lesion of the mitral orifice as would produce, during life, physical signs of mitral obstruction and regurgitation. The disappearance of the presystolic murmur is to be noted. While under previous observation it could readily be demonstrated. It disappeared some time during the three years he was not examined.

I did not see the lad during the last three days of his illness, and am quite unable to state the physical signs in connection with the lungs.

November 8, 1888.

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27. *Case of ulcerative endocarditis.*

Exhibited by Dr. H. M. Fisher.

T. B., æt. 28 years, single, machinist, born in England, was admitted to the Episcopal Hospital November 10, 1888. For the following notes

of the case I am indebted to Dr. George B. Tullidge, the resident physician.

Patient had had one or two attacks of acute articular rheumatism, and for two or three years—perhaps longer—appeared to have suffered from dyspnœa on exertion. Present illness has lasted two months, during which time he has had pain and stiffness in various joints, and fever, and has scarcely left his bed at all.

Upon his admission to the hospital, patient had an anxious expression, dyspnœa, and cough with frothy mucous expectoration; joints reddened and somewhat painful on pressure; pulse very rapid and feeble; numerous moist râles over both lungs; first sound at apex appears roughened; no murmur detected at the base; apex-beat in sixth space to left of nipple; pain on pressure over præcordium; there was fever. Ordered sodii salicylat., gr. x, every three hours.

November 12. Complains of pain over præcordium. Respiration 40, pulse extremely weak; great dyspnœa. For the relief of the pain morphiæ sulph., gr. $\frac{1}{6}$, was given hypodermically, and an ice-bag applied to præcordium. Expectorates blood-stained frothy mucus. Râles more numerous.

14*th*. Delirious. Temperature just before death rose to 107° F. Died in the afternoon.

Post-mortem examination: Right ventricle dilated and contains black and chicken-fat clot. Tissue of heart soft and friable, ventricular walls rather thin. Patch of atheroma on one of the tricuspid leaflets. Left ventricle dilated and empty. Mitral valve contains chicken-fat clot, with atheromatous patches, but no ulcerations. Aortic cusps show vegetations encroaching from $\frac{1}{4}$ to $\frac{1}{2}$ inch on valve orifice. Each of the cusps perforated by jagged ulcerations, of which the largest has a diameter of $\frac{3}{8}$ inch. Aorta above cusps shows numerous deposits of atheroma. Lungs congested and somewhat emphysematous. Right kidney showed on its upper border an old infarction two inches in diameter at its base.

November 22, 1888.

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28. *Arterial degeneration; cardiac hypertrophy; Bright's disease.*

Exhibited by Dr. H. M. Fisher.

J. W., colored, æt. 47 years, married, by occupation a brick-maker, was first seen by me about the middle of last April. Stated that he had suffered off and on from subacute articular rheumatism for several years

past, and that he had Chagres fever on the Isthmus, but, further than this, he did not appear to have had any severe illness. One year ago he began to suffer from dyspnœa, and had, at times, a good deal of cough without much expectoration. Has never spit blood. Suffers a good deal from palpitation. Has lost twenty pounds in weight in the past year. Has orthopnœa at night, and sweats much.

The apex beat is in the seventh space, extends $5\frac{1}{2}$ inches to the left of the sternum. There is a loud murmur, apparently diastolic in time, heard at the second right costo-sternal articulation. No murmur heard over any other portion of the præcordium. Radials very hard; marked *arcus senilis*. Right carotid pulsates very strongly, much more strongly than left. Chest free from râles. Micturition frequent. Œdema of the feet occurred for the first time three days ago. Ordered belladonna plaster to præcordium. Tr. digitalis, gtt. xx. twice daily, and sodii iodid. gr. v. t. d. Pulse 104 and a little irregular.

April 25. Pulse 72 and regular. Feels more comfortable. Continued other treatment and ordered liq. potass arsenit. gtt. v. t. d.

May 1. Complains of pain along course of œsophagus and aching at epigastrium, which is worse when stomach is empty. Stopped iodide and ordered bismuth subnitr., sodii bicarb., āā. gr. x., and pulv. rhei. gr. ss. t. d. a. c.

9*th*. No more pain, but "fluttering about heart," and dyspnœa on exertion. Continued liq. potass. arsenit. and ordered mist. Basham. c. strychniæ sulph. gr. $\frac{1}{48}$ t. d.

22*d*. "Fluttering" continues, and discomfort after eating complained of. Stopped Basham's mixture and ordered acid and pepsin after meals.

30*th*. Much dyspnœa two or three nights ago. Ordered nitro-glycerin in tablet form, gr. $\frac{1}{100}$ t. d.

June 8. Nitro-glycerin, gr. $\frac{1}{100}$ six times daily.

17*th*. Nitro-glycerin gives a good deal of relief to the dyspnœa. Pulse 80 and full, but at about every twelfth beat there is an imperfect systole.

July 7. Not so much palpitation, and no more epigastric pain. Says that he feels better than he has done for six months. Can now sleep for several hours at a time, and with his head low. Continued nitro-glycerin gr. $\frac{1}{100}$ six times daily, liq. potass. arsenit. gtt. v. t. d., and tr. digitalis, gtt. x. twice daily.

24*th*. Has been able to do some work since last note. Has dyspnœa on walking up hill, but none while walking on a level. Pulse 68. Severe night-sweats complained of. Treatment continued.

September 26. Says that he had discontinued the nitro-glycerin for two or three weeks and did not feel so well. Had a bad attack of "asthma"

last night. Some pain in abdomen. Apex beat in seventh space, about three inches to left of the nipple. Very great pulsation of the arteries in the neck. Ordered nitro-glycerin, gr. $\frac{1}{100}$ every four hours, tr. digitalis, gtt. x. every four hours till twenty doses have been taken, mist. Basham, ʒii., c. strychniæ sulph. gr. $\frac{1}{48}$ t. d.

October 1. Was not able to take the prescribed amount of digitalis, the first dose having occasioned dizziness and the second nausea and vomiting, but found that he was able to take 30-drop doses without discomfort. Pulse 84 and more regular, and palpitation has been less marked. Continued tr. digitalis, gtt. xxx. four times daily for three days.

4*th.* Pulse about 80. Tr. digitalis, gtt. xx. twice daily. Severe attacks of dyspnœa being still complained of, ordered nitro-glycerin gr. $\frac{1}{100}$ to be repeated in fifteen minutes when attack of dyspnœa came on, and again in half an hour if necessary, also chloroform inhalations, if nitro-glycerin failed to give relief.

14*th.* Was again relieved by the nitro-glycerin.

17*th.* Again much dyspnœa. Tr. digitalis, gtt. xxx. every four hours, and morphiæ sulph. gr. ¼. Atropiæ sulph. gr. $\frac{1}{100}$ at night.

November 4. Nitro-glycerin for the last two weeks has failed to give relief to dyspnœa. Much œdema of feet and legs. Ordered caffein citrat. gr. v., and sodii benzoat. gr. x. every four hours.

5*th.* Tr. strophanthi, gtt. x. t. d.

9*th.* Pain complained of in right hypochondriac region. Ordered hydrarg. chlorid. mitis, gr. j., sodii bicarb. gr. x. every three hours, at the suggestion of Dr. F. P. Henry, who had kindly visited the patient for me during my absence from the city for a few days.

11*th.* Vomiting and purging last night; very restless; some vomiting of blood. Very strong pulsation at episternal notch. The murmur appears to be more systolic than diastolic and is not transmitted at all to the back or to the arteries in the neck. Œdema has increased very much and dyspnœa very great. Urine cloudy, straw-colored, sp. gr. 1009, highly albuminous; under microscope, granular casts and urates in excess.

12*th.* In attempting to return from adjoining room, patient fell down this morning and died in a few minutes.

14*th.* *Post-mortem examination, fifty-three hours after death.*—Both lungs intensely congested posteriorly, the lower lobe of the right lung being splenified, and portions of it sink when placed in water. The heart weighed two pounds eight ounces before the clots were removed, and one pound fourteen ounces after they were removed. Dr. A. V. Meigs,

who assisted me at the autopsy, found that, on applying the hydrostatic test to the tricuspid valve, the hose being introduced into the pulmonary artery and a stream of water being directed in this way against the tricuspid leaflets, there was absolutely free regurgitation through this valve. The mitral valve, tested in the same way, proved to be competent, and the aortic valve showed a very slight leak. The tricuspid valve admitted the passage of five fingers, while the mitral received four. The left ventricle was greatly dilated, and its walls were hypertrophied, being three-quarters of an inch thick at their thickest part. The right auricle was also greatly dilated, and its wall was in one place half an inch thick. The right ventricle also much dilated and hypertrophied.

The *aorta* was much dilated, and showed numerous atheromatous patches. There was also an atheromatous patch in the anterior leaflet of the mitral valve. The coronary arteries were enormously dilated, being three or four times their normal size. The corpora arantii were enlarged, but there was no marked roughening of any of the valve surfaces and no evidence of valvulitis, old or recent.

Both kidneys were irregular in outline and showed marked diminution in their cortical portions. Under the microscope the Malpighian tufts were found to be contracted, and there was a marked proliferation of connective tissue between the tubules. The walls of the renal arterioles were greatly thickened. The tubular endothelium was highly granular.

The stomach was considerably dilated, but there was no very evident contraction of its pyloric orifice. Its mucous surface was roughened and showed patches of intense congestion. Under the microscope there was seen to be some increase in the mucous connective tissue, and very marked thickening of the walls of the smaller bloodvessels.

The tissue of the liver was unduly tough, and the microscope showed connective tissue proliferation between the lobules and thickened vessel walls.

Microscopic examination of the splenified portion of the lung showed very great distention of the alveolar capillaries, infiltration of the alveoli with epithelial cells and mucous corpuscles, and numerous small infarcts.

The abdominal aorta showed atheromatous degeneration of a very high grade.

This case, though presenting nothing unusual either in its clinical history or in its pathological changes, might still, I think, in the light of comparatively recent investigations, open an interesting field of study. Here we have had degenerative changes in the arterial walls, of obscure origin, evidently antedating cardiac and renal changes. Diminished

elasticity in the vessel walls has unquestionably caused such an obstruction to free blood-flow as to necessitate more energetic efforts on the part of the heart muscle to overcome the resistance.

As long as simple hypertrophy lasted, the heart was quite equal to the increased effort, and the patient experienced no distress and was able to attend to his ordinary work. As soon, however, as the heart became markedly dilated, dyspnœa on exertion was experienced.

With increasing dilatation of the right heart the tricuspid leaflets became, little by little, more incompetent, and systole of the right ventricle became rapidly more and more incomplete. As Dr. Meigs observed, it was not likely, judging from the appearance of the ventricles, that they had been able to empty themselves of blood for several months prior to the patient's death.

The clinical history, too, is instructive on this point. At first the administration of digitalis relieved the dyspnœa, and caused a marked steadying of the patient's pulse. Later, or at least a month before his death, it absolutely failed to give any relief, the dilatation being then so great that no power on earth could prevent the free regurgitation at each systole. At first, too, nitro-glycerin gave relief by dilating the arterioles; after a time, however, perhaps in consequence of increasing degeneration of their walls, no reaction followed its administration, and stasis in the pulmonary circulation and resulting dyspnœa became more and more marked. That nitro-glycerin *is* capable of giving very great relief, even in cases of pronounced cardiac hypertrophy, so long as the dilatation has not become very great, my experience in this case would seem to prove.

How far the kidney changes can be held responsible in this case for the cardiac hypertrophy it would be difficult to say. I think, however, that the arterial degeneration was the primary change. Imperfect depuration of the blood may, perhaps, by causing spasm of the arterioles, have interfered still further with the circulation and thrown more work upon the already overburdened heart and increased the mischief.

Dr. Meigs said Dr. Fisher was good enough to ask me, upon one occasion, to see the man from whose body the specimens that have been exhibited were removed, and I had the advantage, besides, of being present at the autopsy. The case was one of interest from several points of view. When I examined the patient, no murmur, or only the faintest approach to one, could be heard at the aortic cartilage, but the moment the stethoscope was moved an inch to the left, being then over the body of the sternum, a very harsh murmur became audible, which was still louder over the lower part of the sternum and had its point of maximum

intensity at the left edge of the ensiform cartilage in the tricuspid region. The time in the cardiac circuit occupied by the murmur I could not satisfactorily determine, as the heart's action was somewhat rapid and feeble, but I was entirely satisfied that it was not diastolic, that it had not the rhythm of an ordinary typical aortic regurgitant murmur. This murmur was inaudible at the apex of the heart. The aortic flaps and opening present, as may be seen, little alteration, except that both are much enlarged and that the corpora arantii are at least three or four times the natural size. When water was poured in the aorta, the flaps approximated well and closed the opening entirely, except for a leak directly in the centre, where the enlarged corpora arantii prevented perfect closure, and a stream the size of a pin flowed backward into the ventricle. When a tube was introduced into the aorta, passed beyond the valve, and a stream of water projected into the ventricle, the mitral valve was seen to close perfectly and to be entirely water-tight. When the same experiment was performed upon the right heart, the tube being thrust into the pulmonary artery beyond the apparatus of the valve, and the water turned on, it was found that the valve flaps were so entirely incompetent to close the dilated opening that the whole stream of water from an ordinary tap would flow backward through the opening. This experiment, which I look upon as a valuable test in cases of suspected incompetence of the mitral or tricuspid valves, and one which, unfortunately, is very seldom tried at post-mortem examinations, demonstrated entirely to my satisfaction that the view I took and expressed during the life of the man, that the murmur was not due to aortic regurgitation, was correct. The murmur, I believe, was one of tricuspid regurgitation, and I failed to make a positive diagnosis to that effect because of the rapidity and feebleness of the heart's action. There could have been, during life, only the slightest amount of aortic regurgitation, and very likely none at all, as it is probable that, with a firmer pressure within the aorta than could be produced by the weight of a column of water an inch or two high, the valves would have been found entirely tight.

The question of the point of origin of the disease in this case is one of exceedingly great interest, and it opens for discussion a wide field in the train of symptoms that we call Bright's disease. Goodhart has well said, "Old age is not an entity, but a set of conditions predisposing to that state which is called chronic Bright's disease," and the saying that "a man is as old as his arteries" should become almost an aphorism with us. We are tending, I think, rapidly of late years, toward a more correct appreciation of this condition, the first step, or one of the first steps, in the right direction having been made by Gull and Sutton in

their classic article upon "Arterio-capillary Fibrosis." Fothergill's recent little book upon *Vaso-renal Change versus Bright's Disease* is an excellent résumé of the present state of knowledge upon the subject, but there is little that is original in it, except his pet theory in regard to what he calls the "uric-acid reversion," which will hardly, I think, bear close examination, for, if there is an organ in the body which seems ordinarily the last to undergo organic change in this condition, it is the liver.

The case Dr. Fisher has described would, a few years ago, in the absence of any special attention having been called to the renal condition, have been unhesitatingly put down as one of primary organic disease of the heart, and we would have carefully endeavored to elicit a history of rheumatism, which complaint has in the past been made accountable for a vast amount of cardiac mischief that was in no way due to it. Now, however, I think there need be little hesitation in asserting that the extensive and widely-diffused changes which are found in the arteries and arterioles in such cases, and which were found in this case, long antedated the existence of other organic changes in the heart or kidneys.

In the course of my studies at the Pennsylvania Hospital during the last six or seven summers, there are several things that have struck me in connection with my cases of Bright's disease, and although some of these things may be well known to the members of this Society, yet perhaps it will not be amiss to make brief mention of the more important ones.

In the first place, I have come to believe that there is no one symptom or set of symptoms necessarily connected with the disease; that the earliest sign to which our attention may be directed, or the first thing the patient has complained of as having made him aware that he was no longer in perfect health, varies to any extent in different cases, and seems to depend upon what particular organ is first injured by the changes of the arteries and arterioles—sometimes the earliest symptoms will draw attention to the stomach, again to the lungs or nervous system, or it may be the heart or kidneys that will first present the signs of the functional disorder, which will later be followed by signs that will force upon us the knowledge that our patient has grave organic disease. This statement is contrary to what is now very commonly taught, for so lately even as in the number of the *Medical News* of Dec. 3, 1887, there is a report of a lecture upon chronic Bright's disease, by James T. Whittaker, M.D., in which he makes the dogmatic statement that "a disturbance of digestion characterizes, more or less, every case of Bright's disease."

Second, it has surprised me to find how little structural change there

is of the kidney itself, in cases even in which the patient has died with a full development of all the symptoms that are ordinarily considered as typical of the complaint. So much is this the case that I have come almost to believe that it is quite rare for men to die solely as the result of a chronic destructive process of the kidney which finally reduces the amount of secreting substance, but that it is inadequate to carry on the necessary excretory functions of the body, and that in the cases in which we have been in the habit of attributing death to this cause it has been in truth brought about by other, and at present inexplicable, things.

Third, in cutting sections of the heart of a woman who died of Bright's disease last summer in the Pennsylvania Hospital, I found in the fat, a thick layer of which covered the anterior surface of the organ, an extensive network of muscular fibres running far out from the common mass of the muscle. These muscular fibres were undergoing brown atrophy, or fatty degeneration. Upon examining sections that I have in my possession from other hearts whose anterior surface was covered with a layer of fat, I found here and there scattered through the fat in them also remains of what appeared to be degenerated muscle. Would not this seem to show that the fat grows somewhat at the expense of, and consonantly with a decrease of the amount of muscle?

Lastly, the condition that I have found most constantly and most extensively developed has been the following appearance of the arteries and arterioles: upon examining cross-sections, there is seen in the vessels, lying inside the wavy fenestrated membrane, which in the natural condition has in the smaller vessels little inside of it except the endothelial cells, crescents of a hyalin or granular material: or this material may form a ring of even thickness around the whole vessel, but still lying inside of the wavy line. The change is plainly one of the inner coat of the vessel, and not one of either the muscular layer or of the adventitia, as we are commonly told is to be found in such cases. This I have seen in the vessels of the brain, of the lungs, heart, spleen, kidneys, and in the larger arteries. When we turn back to consider the way in which this train of symptoms was originally discovered, that at one time men were treated for the disease dropsy, that then it was observed that in dropsy there was commonly albumin in the urine, and next Bright noticed that when there was dropsy and albuminuria there were commonly found diseased kidneys, it is no wonder that we have gone somewhat astray and have looked upon the kidney as the prime source of disease, and have spoken of the secondary effects produced upon the heart and other organs, when in truth the structural changes in the heart may as often antedate those in the kidney as the contrary.

There is much more in connection with this most interesting topic and with Dr. Fisher's case that might be mentioned, but I have already sufficiently long occupied your time. *November* 22, 1888.

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29. *Partial fusion of the two segments of the pulmonary semilunar valves.*

Exhibited by Dr. W. Osler.

The specimen was removed from a woman who, so far as was known, had not suffered from any symptoms of heart disease. One of the pulmonary cusps was normal, and the other two had partially united, so that from the ventricular side only one valve was apparent, with a notch at the edge, indicating the point of separation of the two portions. From the arterial side there were two sinuses of Valsalva, but the median *raphé* extended only a short distance up the conjoined segment. The edge of these united cusps was a little thickened, and the valve did not look as if during life it could have been competent. The pulmonary artery itself was not atheromatous.

Dr. Osler held this condition of fusion of the segments was, in the majority of instances, congenital. In his experience they were more common in the aortic than in the pulmonary valves, but he had seen within the past two years three other specimens of this anomaly of the right side. *November* 22, 1888.

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30. *Pneumonia of the right middle lobe; ulcerative endocarditis; purulent meningitis.*

Exhibited by Dr. J. C. Wilson.

D. R., æt. 25 years, an Italian laborer. Admitted to the Men's Medical Ward of the Philadelphia Hospital, October 28th, 1888. No history was obtained. Patient had been ill, with the signs of plastic pleurisy of the right side and subfebrile temperature for some time. There was a slight purulent discharge from the left ear. The febrile movement suddenly became high, eleven days before death, with the symptoms and physical signs of a right-sided pneumonia. The type of the fever was irregular remittent, with abrupt exacerbations and remissions varying from 3° to 8° or 9° F., in the course of a

few hours. There was slight cough, and scanty expectoration; for thirty-six hours prior to death there was delirium and slight opisthotonus; pupils not affected. Patient moderately emaciated; no œdema; no infarcts on exterior nor ecchymoses; face unusually pale and extremities bloodless.

Liver unusually large, weight $6\frac{1}{4}$ pounds; capsule normal; markings of acini distinctly seen through capsule on section, dark-red and congested; acini unusually large; central veins not markedly engorged; appearance that of a simple hypertrophy of the liver; no fatty nor any low change.

Biliary passages, stomach, and small intestine, all normal; considerable colitis in large bowel, without ulceration; mucous membrane covered with mucus; mesenteric and retroperitoneal glands normal.

Spleen enlarged; weight two pounds five ounces; large and recent infarct (culture made from it).

Bladder normal.

Kidneys large; congested; contained large hemorrhagic infarcts, recent thrombi being detected in some of the vessels; one infarct was confined to the medullary portion.

Peritoneum normal; no ulceration, nor point of suppuration.

Lungs: Rather recent plastic pleurisy over upper and middle lobe of right lung; middle lobe of right lung gave evidence of a recent pneumonia; no pulmonary infarcts; some bronchitis, but no ulceration in bronchi; bronchial glands normal.

Heart: Pericardium normal; heart contained clots in all the chambers. No evidence of change in myocardium. Aortic, pulmonary, and tricuspid valves normal. Near free borders of auricular surface of mitral leaflets three circumscribed oval areas of ulceration. Of these one occupies anterior leaflet, and presents the appearance of actual loss of substance, except at its edges, which are infiltrated and elevated above the level of the surrounding surfaces. This ulcer is in size 1 by 1.5 cm. Upon the corresponding surface of the posterior leaflet there is a patch of recent, soft, exuberant vegetations, the base of which is of about the same size, and near it a similar smaller patch.

Aorta normal.

Brain: Extensive purulent meningitis, with purulent fluid in ventricles. Brain substance normal.

Middle ear was opened, but no suppuration had occurred so far as could be detected. *November* 22, 1888.

31. *Ulcerative endocarditis.*

Exhibited by Dr. H. F. Formad.

The person from whom this specimen was obtained was a colored man, who had been complaining of slight ill health for a few weeks before his death. His indisposition seemed slight, and he paid little attention to it, going about his daily occupation as usual. While eating in an oyster-house he dropped dead. The autopsy showed extensive acute ulcerative endocarditis. The valves of the left side of the heart all contained ragged ulcers, and were thickly studded with vegetations. One of the mitral leaflets was extensively lacerated, probably the immediate cause of death. In the muscle of the left ventricle was a hole, into which the end of the finger could be thrust with ease, the result of the ulcerative process. The lesion was so recent that no aneurismal dilatation of the wall had commenced. The myocardium was full of miliary aneurisms, probably the result of embolism. *December* 13, 1888.

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32. *Extensive calcareous deposit in the pericardium.*

Exhibited by Dr. E. T. Bruen.

Post-mortem notes on William D., æt. 59 years, male, white. The body was medium-sized, well nourished; small scars on legs.

Thorax: General adhesions of the pleura; the layers of the pericardium were universally adherent; over the right auricle anteriorily and over the posterior part of the left ventricle there was an extensive calcareous plate. This was unusually dense, hard, and over five inches broad, varying in thickness from a quarter to three-eighths of an inch. The anterior wall of the left ventricle was free. The left ventricle was normal in size; the right ventricle somewhat dilated; the left and right auricles very markedly dilated. The valves of the heart were normal, but the pulmonary artery was dilated to more than double its natural calibre; there were also one or two small patches of atheromatous deposit in the endoarterium.

Lungs: In the left pleuræ there were numerous old adhesions, in places infiltrated with serum. Extensive recent pneumonia of the posterior lateral portion of the upper lobe; lower lobe was crepitant.

Right lung crepitant throughout, but congested and œdematous at lower part.

Kidneys were large, coarse-looking, containing numerous cysts. Capsules not adherent. Spleen was large, capsule firm, with numerous fibroid changes.

Liver showed extensive monolobular sclerosis.

Clinical notes.—These are unfortunately negative. The patient was received into the Philadelphia Hospital from the almshouse, and died of exhaustion within a few days after admission. Such inquiry as was possible elicited that no marked symptoms of cardiac disturbance had been observed. *February* 14, 1889.

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33. *Heart with congenital malformation of the mitral valve.*

Exhibited by Dr. E. T. Bruen.

There was an adhesion between the anterior and posterior leaflets of the valve which, from the manner in which the chordæ tendineæ were inserted, was evidently congenital. This afforded an obstacle to the direct passage of the current of blood, which found its way into the ventricle to one side of the obstruction. It was doubtful, however, that any real obstruction at the valve existed. On the ventricular aspect of the leaflets there were evidences of recent endocarditis. The aortic valves were shrivelled and thickened, and the aorta in an atheromatous condition. The kidneys were cirrhotic. *February* 14, 1889.

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34. *Aortic stenosis.*

Exhibited by Dr. A. A. Eshner.

Nancy M., æt. 90 years, was admitted to hospital March 25, 1889, with shortness of breath, palpitation of the heart, and muscular weakness, which had existed for about three weeks, and œdema of the lower extremities, which had been noticed only for four or five days. Her most marked symptom was orthopnœa. Patient was in the house for a few days, but a careful physical examination was not made. The urine contained a small quantity of albumin and hyaline and granular casts.

The heart was noted to be arhythmical, weak and slow, and the pulse of variable strength. Four days after admission, the patient suddenly fell over and died. At the autopsy the pericardium was found to contain a slight excess of clear, straw-colored serum.

The heart was greatly enlarged. The right cavities contained a large amount of clotted blood. The left ventricle was empty. The tricuspid orifice was dilated, admitting four fingers and the thumb. The valves were normal. The cavity of the right ventricle was dilated. The mitral orifice was slightly contracted, admitting the tips of three fingers. The valves were thickened, and the chorda tendinea attached to the posterior papillary muscle was greatly thickened, rigid, and contracted. The left ventricle was somewhat dilated, with irregular thickening of the muscular wall. The aortic orifice was but a mere chink, which permitted of enlargement upon gentle pressure by the finger. There were but two cusps, as if two of the leaflets had been merged into one. Both were excessively thickened, and contained hard masses of calcareous deposit, which was the more abundant in the posterior sinus. The valve appeared fairly competent, permitting of but a small reflux of fluid. The ascending aorta was roughened by atheromatous changes.

Both pleural cavities contained large quantities of somewhat blood-stained fluid, which had compressed the lungs, both of which were small, but crepitant throughout.

The kidneys were small and contained numerous cysts of retention. The capsules were thickened, but stripped readily, leaving behind a markedly granular surface. On section, the cortex presented the same granular appearance seen superficially, and was much diminished.

April 11, 1889.

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35. *Hemorrhagic pericarditis.*

Exhibited by Dr. A. A. Eshner.

Mary M., æt. 73 years, was admitted to the skin ward February 15, 1889, with a history of a fall and injury to the left leg, followed by reddish-purple discoloration and swelling. The heart-sounds were feeble, but at the apex and at the ensiform cartilage a blowing, systolic murmur was heard. Thought to be a case of erysipelas, she was sent to the ward for the treatment of such cases. Under the application of a bandage, and with rest in bed, the swelling and discoloration readily diminished. The cardiac murmur persisted. With the occurrence of

other cases of a somewhat analogous nature, a diagnosis of scorbutus was made. The temperature at no time rose above 100°. The woman's mental condition was such that she refused to take nourishment, and on April 3, 1889, she finally died of inanition and exhaustion.

At the autopsy the pericardial cavity was found to contain six ounces of bloody fluid. The parietal and visceral layers of the pericardium were adherent in many places, and the heart was covered by an irregularly distributed layer of lymph, evidently of not very remote origin. The walls of the organ were made up in part of a considerable layer of fat, the muscular tissue in places being extremely thin. The valves and orifices showed no marked changes. There was considerable thickening of the mitral leaflets, and there was a fenestrum in one of the aortic cusps.

The kidneys were small and contracted. The capsules were slightly thickened, but stripped with moderate ease, leaving behind a somewhat granular surface. On section, there was evident diminution of the cortex. *April* 11, 1889.

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36. *A case of malignant endocarditis.*

Exhibited by Dr. C. Walter.

Charles O., white, æt. 30 years, native of France, unmarried, was admitted to the Nervous Wards of the Philadelphia Hospital on December 15, 1888, complaining of partial right hemiplegia, difficulty in speech, and an exhausting diarrhœa.

His previous history was as follows: Syphilis six years before and rheumatism one year prior to admission. Had indulged excessively in alcohol. He had previously been in the hospital complaining of some intestinal disorder. In November, 1888, about three weeks previous to his last admission to the hospital, he was one morning suddenly stricken with a complete right hemiplegia and aphasia. He did not lose consciousness.

On admission he had almost entirely regained power in the arm and leg, the face remaining slightly affected, and the speech at times indistinct. His nervous condition and its causative lesion has already been reported to you by Dr. Mills, and I therefore will not do more than thus mention it. He was at the time very much prostrated, was anæmic, emaciated, with clubbed fingers, slightly œdematous feet, had headache,

frequent and profuse epistaxis, dyspnœa on exertion, cough with mucopurulent expectoration, and a severe diarrhœa: the stools being liquid, yellow, with white particles. Temperature 100°; pulse 116, weak and water-hammer in character; respirations 34.

Examination revealed very loud, blowing heart murmurs, systolic and diastolic in turn, over both the aortic and mitral regions, propagated in the usual directions and heard with varying intensity all over the chest. The heart was enlarged, impulse was forcible, action rapid and irregular. Lungs were slightly congested, with evidences of bronchitis. Spleen enlarged. Liver normal. Examination of urine gave signs of cystitis.

After a few days his condition seemed to improve, but the temperature fluctuated irregularly between 97° and 102°, as shown by the chart, being generally higher at night, at times becoming normal for a few days, only to reassume its somewhat hectic type without apparent cause. The congestion of the lungs and œdema of the feet disappeared. Urine contained albumin and hyaline and pale granular casts which persisted but a week. The diarrhœa remained unchanged throughout the attack, averaging three or four stools per diem of the character described.

January 5. Patient complained of pain and discharge from the right ear, which proved to be due to an abscess of the middle ear.

On the 14th he had a chill, and his temperature rose to 102°, continuing to rise until it reached 104° on the 15th. There was marked dyspnœa, the respiration becoming Cheyne-Stokes and ranging from 28 to 48 per minute. Pulse 120, very weak and irregular. Death occurred on the night of the 15th.

Autopsy, thirty-two hours after death.—The condition of the brain has been reported. There was softening of the motor region of the left hemisphere involving a portion of the Island of Reil.

Thorax: Pleura normal. Lungs congested throughout. At base of upper lobe of left lung there was early red hepatization extending about three inches vertically and entirely across the lobe.

The *pericardium* contained about f℥j of clear serum. The heart was hugely enlarged. Chicken-fat clots distended both auricles and were found in the ventricles. Walls of both ventricles much hypertrophied and their cavities dilated. The pulmonary and tricuspid valves normal. The aortic leaflets had large vegetations, formed of substrata of limy material covered by fresh, fibrinous deposits. On the inner surface of the aorta, and just above the upper edges of the sinuses of Valsalva, were found three small ulcers showing distinct loss of substance. The

aorta was somewhat atheromatous. Mitral valves a little thickened, but no fresh vegetations.

Abdomen: Intestines congested, spleen large and soft, liver somewhat cirrhotic and congested. Kidneys were swollen, dark-red, and congested; capsule stripped readily. There were no infarcts.

I report the case not because of anything unique or very unusual in its symptomatology or course, but because it is another specimen of that endocardial lesion which is, even yet, so frequently mistaken for other and less surely fatal diseases. *May* 9, 1889.

IV. THE ORGANS OF RESPIRATION.

1. *Cases of tubercular pleurisy.*

Exhibited by Dr. W. Osler.

Apart from the pleurisy which so constantly accompanies phthisis, we have, first, an acute tubercular pleurisy, identical in its clinical features with the sero-fibrinous form, which is illustrated by the following cases:

Case I. *Tuberculous pleurisy.*—James K., æt. 60 years, admitted June 4, 1887. Had been ill three or four weeks with cough and soreness in chest. No history of consumption. Of seven brothers, six are living; of six sisters, three or four have died. When examined on the 10th there was dulness over the right chest, from the second rib in front and the spine of the scapula behind; dry tubular breathing; a few râles when he coughed; tactile fremitus diminished; voice sounds loud and articulate; the dryness and intensity of the breath-sounds are remarkable; the heart is not dislocated. Pulse 84; respirations 28. I did not see him after the 21st. On July 19th he was tapped, and two pints of blood removed. He remained in bed through August, getting very pale and weak. On September 20th the examination gave the following results: Dulness from the third rib on the right side, and from the spine of the scapula. Tactile fremitus present, not increased; about the same as on the other side. Distant, but distinct blowing breathing over whole back, most marked toward the posterior axillary fold; on coughing, dry râles; voice sounds intensified; apex-beat at the left nipple; is very anæmic and cachectic. Fluid was determined by hypodermic puncture. Cough, no expectoration. He gradually sank, and died October 8th. The temperature throughout ranged from 98° to 190°. Occasionally the evening temperature was 101° or 102°.

Post-mortem: Left pleura small amount of fluid, adhesions at apex; right pleura sacculated, contained about two quarts of turbid fluid. Left lung, hypostatic congestion at base; a few caseous and calcareous masses at apex; right lung compressed, only antero-lateral margin was crepitant; at apex an aggregation of nodular tubercles, some of them calcareous. Pleural layers much thickened; many bands pass across the

cavity and separate it into irregular sacculi; in both pleural layers there were numerous small tubercles; no tubercles in other organs. Slight contraction of kidneys.

Case II. *Cirrhosis of liver; acute tuberculosis of lungs.*—E. M., æt. 47. Admitted on August 25, 1887. Family history good; was well up to the middle of July; gradually became jaundiced, and ascites developed. On September 22d he had a profuse hemorrhage from the stomach, and also passed blood by the bowel. On the 23d friction was noticed in left axilla, and there were numerous râles at the bases. He gradually sank, and died on the 26th.

The autopsy showed extreme cirrhosis of the liver, effusion in peritoneum. No erosions in stomach. Both lungs presented many fresh tubercles, the size of split peas. At apices a few nodular caseous masses. Left lung was collapsed. Three or four pints of effusion in pleural cavity. The pleura covered with flakes of lymph, and on both surfaces many fresh tubercles. There was a good deal of hemorrhagic effusion between and beneath the fibrinous exudation.

In both of these cases there were foci of old disease in the lungs. In the patient with cirrhosis it is interesting to note that the acute tubercular process supervened at the close of an exhausting illness. The opinion is gaining ground, particularly in Germany, that very many of the instances of ordinary pleurisy are tubercular in character.

Second: Chronic tubercular pleurisy, which is usually of an adhesive variety, producing thickening of the layers. It may be uni- or bi-lateral, and is not infrequently found accidentally in death from other causes. A case of this kind occurred ten years ago in the University Hospital, in a robust girl of twenty-three, who died of malignant scarlet fever, after three days' illness. The right pleura was enormously thickened, the layers adherent, and between them numerous tubercles of all sizes. The following cases illustrate the occurrence of this condition in persons the subjects of chronic affection of the cerebro-spinal system:

Case III. *Tuberculous pericarditis and pleuritis.*—Benj. G., æt. 61 years, an old hemiplegic, died somewhat unexpectedly, after a few days' illness, in the nervous wards. An apoplectic cyst was found in the right lenticular nucleus and the adjacent part of the internal capsule. The pericardium was greatly thickened, particularly the upper part over the auricle and great vessels. Universal adhesion of the layers, the combined thickness of which was in places as much as from $\frac{1}{4}$ to $\frac{1}{2}$ inch. Tubercles were scattered through both layers, and here and there large caseous masses. Both pleuræ adherent; no effusion; an eruption of fresh tubercles on visceral layer, small and gray;

a few scattered tubercles through the lungs; no cavities; no cheesy masses. The bronchial glands contain a few nodules.

Case IV. *Tuberculous pleurisy.*—John G., æt. 38 years, also from nervous ward, with chronic sclerosis.

Post-mortem: Body emaciated; a few cutaneous hemorrhages; in thorax universal adhesions on the right side, on the left two fibrous patches on pleura. The right lung was crepitant throughout, except at apex, where there was an old cavity, the size of a marble, and several small caseous masses in fibroid indurated tissue; no miliary tubercles; parietal pleura thickly studded with miliary granulations; toward the base and anterior edges there were layers of plastic lymph, with slight blood-stained effusion. Both layers of pleura were hemorrhagic, and on visceral leaf the exudation extended into the lung. The tubercles were small, and very thickly set. There was one small nodule at left apex. No tubercles in other organs. *November* 13, 1887.

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2. *Fibrous polypus from the nose.*

Exhibited Dr. H. R. Wharton.

This interesting specimen was removed from the nose of a woman, æt. about 30 years. The growth extended from the posterior nares into the pharynx, and was attached by a narrow pedicle. I should like to call attention to the well-marked pedicle, which greatly facilitated its removal, and which condition, I think, is most likely to be found in the more common cases of ordinary mucous polypus.

February 9, 1888.

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3. *Pulmonary cavity treated by incision.*

Exhibited by Dr. E. T. Bruen.

Post-mortem notes on Alois T., æt. 27 years, Swiss. Admitted to Philadelphia Hospital September 22, 1887. Died January 2, 1888. External appearance: medium-sized man, moderately well nourished. Feet and ankles œdematous.

Thorax: Right lung, some adhesions in pleural sac. Lung tissue œdematous, showing extensive bronchiectasis and emphysema, the latter especially at the edges of the lobes. A few tubercles noted in the upper

lobe, and some fibroid induration, with small cavities in middle and upper lobes. These cavities appeared to be old, and due to dilatation of the bronchi, having a distinct and well-defined lining membrane, and communicating freely with the bronchioles.

The parenchyma of the left lung had been largely destroyed, the visceral pleura moulded by adhesions with pleural sac. On section the entire lung substance had been destroyed, and a large cavity occupied the whole space from apex to base. In this cavity nothing but the frame-work of the lung could be seen. This frame-work consisted of fibrous bands and bloodvessels, traversing the cavity in all directions, and with but very little lung structure. The cavity contained about eight ounces of grumous purulent liquid, with fragments of lung remaining in it. The parietal pleura presented a thickening equalling three inches, which was increased somewhat by the atelectatic remnants of the lung adherent to it. Generally, the lung presented the appearance of a chronic broncho-pneumonia, with unusually well-marked bronchiectasis.

The pericardial membranes showed the characteristic lesions of acute pericarditis of recent type. Enormous bilateral heart-clots were found, extending far into the vessels. Heart-valves normal.

Bronchial glands enlarged, but not cheesy. The ninth rib had been resected two inches from its vertebral attachment, and two inches of the rib removed. The upper portion of the cavity was much smaller than the lower portion.

The history of the case was indefinite, but it was gathered that the diease in the lung came on suddenly about six weeks previously, with pneumonic symptoms. At date, October 20th, the patient suffered very much from cough, expectorated freely pus and nummular sputa, which contained tubercle baccilli. The temperature indicated absorption of septic matter; ranging from 100° to 102°. The location of the vomica prevented the antiseptic inhalations from really reaching the part, and the patient could not readily empty the cavity by coughing. After consultation with two of my colleagues, Drs. Curtin and Osler, it was determined to open the cavity and attempt to secure local drainage. The physical signs of superficial cavity being most intense at the angle of the left scapula, it was decided to operate at this point. Resection of a rib was requisite to obtain working-room, as the ribs are close together at this point. On October 27th the patient was placed under ether, and Dr. Janney, assisted by Dr. Porter, removed about one and a half inches of the rib just within a line drawn from the angle of the scapula. The pleural surfaces were found to be adherent, and the pleural membrane was slightly thickened. It was decided to attempt to

enter the cavity with the large needle of an aspirator, which was accordingly introduced. Although the physical signs had been so pronounced, yet the needle was introduced two inches into the lungs in several directions without entering the cavity. The punctures caused no coughing, disturbance of respiration, or, in fact, any inconvenience. When the needle was disconnected from the aspirator some little blood escaped, and sufficient air to extinguish a match. Finally, a large-sized trocar was introduced in two directions, and it was thought, from the manner in which the front of the instrument moved, that the cavity had been penetrated. A small drainage-tube was introduced, and the wound dressed after the antiseptic method. On the following day the dressings were removed, and it was found that at least an ounce of pus had escaped from the sinus. It was decided to enlarge the sinus in which the drainage-tube had been placed by dilating it daily with rubber catheters. The second day after the operation, while this dilatation was being practised, suddenly several ounces of pus gushed through the sinus and escaped externally, and thereafter the sucking was heard, so familiar after an empyema has been incised. It was manifest that the cavity had, at last, been freely opened, for now injection of fluids into the vomica could be coughed up through the bronchi. A blunt probe could be passed seven inches directly into the chest, and the impulse of the heart could be detected along the probe.

There were several points of interest in connection with the details of the operation: the first of these was the fact that the cavity was situated at least two inches from the pulmonary pleura. Secondly, the first two days the cavity was either not entered, or only a minute opening was created, and yet no damage to the lung occurred, and the temperature fell a degree and a half.

The propriety of entering similar cavites with the thermo-cautery has been suggested, but when the cavity is situated deeply may not the mode adopted in this case be more simple? Certainly there were no evil effects in the present case. I would suggest, therefore, the puncture of cavities in the lung by a trocar, and then a small drainage-tube can be introduced through the canula; the drainage-tube can be exchanged for a large one by dilating the sinus. The resection of one or more ribs seems to me an important feature in the operation for the opening and drainage of pulmonary cavities, because retraction of the costal wall is facilitated by this measure. The general condition of the patient has steadily improved since the operation, and at date his temperature is normal, with greatly reduced cough and betterment of the general strength. On November 1st he passed under the care of my colleague, Dr. J. C. Wilson. *February* 23, 1888.

4. *Whooping-cough, with pseudo-membranous laryngitis and long-standing enteritis.*

Exhibited by Dr. A. V. Meigs.

An infant, born in the Preston Retreat September 21, 1887, and brought to the Sheltering Arms October 30th. The child was premature by one month, and always rather puny and ill-nourished, and had had numerous small furuncles. When brought to the institution his condition was found to continue so bad that he was given artificial food twice a day, and as he seemed to improve was, before long, weaned. After some variations in emaciation, he at last improved, picked up a good deal of flesh and throve. There was, however, whooping-cough in the institution, and about February 14th he developed some cough. This went on from bad to worse, so that he had at last great difficulty in taking food, and he died in a fit of coughing on February 25th. There was at no time any laryngeal difficulty in breathing, or stridor, though before death there were râles in the chest.

Post-mortem, ten hours after death.—The brain and spinal cord could not be examined. Body well nourished.

Larynx was covered upon its internal surface with a thin pseudo-membrane, which extended well down into the trachea.

Lungs: The general surface very pale and bloodless, but there were numerous dark spots of splenization scattered here and there, which upon section were seen to penetrate quite deeply into the interior. No areas of true pneumonia.

Heart appeared to be large, and both auricles, especially the right, filled with black clots. The right ventricle distended with dark clots, the left firmly contracted. Organ otherwise normal, and the foramen ovale entirely closed.

Liver natural.

Spleen natural.

Kidneys congested, the cortical portions being red and injected, and this particularly marked at the junction of cortex and medulla, which latter portion was pale.

Stomach contracted, and presented no unnatural appearances.

Mesenteric glands much enlarged, some being as much as from one-quarter to one-third of an inch across.

Small intestine: There were ten invaginations. In some cases the mucous membrane and villi looked natural, and in others the gut

seemed very thin, and the mucous layer lessened in amount. Peyer's patches in some spots very thin and injected, and again injected, thin at the centres, and with raised and everted edges. This was particularly marked in the glands directly above the ileo-cæcal valve.

Large intestine: Its surface was thickly strewn with solitary glands, as large as a small pin's-head, and many of them had a small ulcer upon the summit of the inner surface.

The case is interesting on account of the false membrane in the larynx in the course of whooping-cough, and the absence of any evidence of inflammatory or even congestive condition of the lungs in the disease, these organs being, as stated, more bloodless even than usual. It is further interesting as an exhibitien of the state of the digestive tract in a case of cured marasmus, or rather of one in process of recovery from that complaint. The infant was during November and December in very bad condition, having sunken fontanelles, overriding of the bones of the skull, and great emaciation; but during the last two months of life he improved much, became comparatively fat, and the head rounded very much. The intestinal condition was probably the cause of the malnutrition, and it may also properly be assumed that if it had not been for the intercurrent attack of whooping-cough, always a very fatal disease amongst young infants, he would have eventually recovered. The intestinal condition is doubly interesting, when considered in connection with the other specimens from cases, in many respects parallel, that I have exhibited to the Society in the past few months. If six or eight cases can be considered to show anything, these show what are the appearances to be looked for in ordinary cases of malnutrition, the so-called marasmus of young infants. *March* 22, 1888.

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5. *Tuberculous (?) tumors from epiglottis, with two microscopic sections.*

Exhibited by Dr. S. Solis-Cohen.

I have seen the patient from whom these tumors were removed, and I have been given the principal points of interest in the history of the case by Dr. J. Solis-Cohen.

The case is one of extensive tuberculosis laryngitis—in fact, the most extensive case Dr. Cohen has seen, in the person of a gentleman æt. 45 years, who had tenderly nursed a wife who had died of tuberculosis pulmonum. The disease, as is usual in acute tuberculosis

laryngitis, began in the epiglottis, with infiltration, thickening, and ulceration, which extended first on the left side, as is usual in the disease, and then on the right side, as far down as the vocal bands, eventually greatly destroying the epiglottis, first to the left of the mesian line, and then to the right, and to such an extent that, when last seen, a mere longitudinal strip of epiglottis remained on the left, and a thickened strip in the form of an elongated stump upon the right.

Several weeks before the ulceration had reached this extent, Dr. Cohen removed with forceps several growths, some of which are herewith shown, from the midst of the ulcerating lingual face of the right half of the epiglottis, the largest of which, still covered with intact mucous membrane, is an irregular globular mass, of about three-eighths of an inch in its largest diameter, with a distinct point of implantation.

The disease is now of six months' duration, and is at present in an apparent state of quiescence, cicatrization beginning at the outskirts of the process. The essential topical treatment producing the result consisted in thorough curetting, followed by thorough friction with lactic acid in concentrated solution.

No bacilli were found by me in three examinations of sputum, and none appear in the microscopic sections of two of the smaller growths, kindly made by Dr. Gray, of the U. S. Army Medical Museum. I submit these sections and specimens without description, or expression of opinion, as I desire them to be referred to the Committee on Morbid Growths, to determine whether or not they exhibit a tuberculous histology.

There is no doubt in my own mind, as there is none in that of Dr. Cohen, that the physical signs in this case, both those more recently elicited by percussion and auscultation of the chest, and those originally visible upon laryngoscopic inspection, are positive evidence of tuberculosis.

The absence of bacilli from these specimens, if confirmed by the further examinations to be made by the Committee, give them a peculiar importance, beyond that which in any event attaches to tuberculous tumors of the larynx. *April* 12, 1888.

Report of the Committee on Morbid Growths: "Your committee reports that the sections reveal the surface of the growth covered with pavement epithelium, and the centre composed of granulation tissue and giant-cells, with peripherally-placed nuclei. Tubercle bacilli, although

carefully sought for, could not be demonstrated. Control stains in a tuberculous lung, made in the same fluid, show the bacilli very freely present."

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6. *Myxomatous degeneration of myrtiform tumor of cavernous tissue from posterior extremity of left middle turbinated body.*

Exhibited by Dr. S. Solis-Cohen.

This specimen is exhibited for its rarity. I am not aware of any like it on record, though I have not made so careful a search into literature as would warrant a positive statement that it is unique. My impression is, that careful examination of the numerous mucous polypi removed daily would furnish quite a fair array of comparable specimens. The interesting feature is, that externally and rhinoscopically, closely resembling the ordinary "posterior nasal hypertrophy"—as the loose nomenclature of the day has it—microscopically the specimen shows an outer layer of nearly intact mucous membrane, slightly thickened, enclosing a typically myxomatous mass, in which the venous channels of the erectile tissue can still be distinctly made out in circular transverse section, their walls being here and there infiltrated with small inflammatory round-cells, and generally a few fibres of connective tissue remaining in their neighborhood. The specimen thus appears to be a circumscribed "hypertrophy" on its way toward polypdom.

The patient is a gentleman of some forty or fifty years of age, who had long suffered from asthmatoid paroxysms of dyspnœa, and severe paroxysmal cough, attributed to phthisis. When he came under Dr. J. Solis-Cohen's care, it was discovered that his nasal passages were full of polypi, of which some thirty odd were removed. They varied in size from that of rice-grains to that of a small white bean, and were mostly strung along the inferior surfaces of the middle and superior turbinated bodies of both sides, especially the left, resembling a row of pearls in the shell.

The specimen presented is not one of those pearl-like polyps, but appeared as a myrtiform mass, somewhat lighter in color than the neighboring mucous membrane, on the posterior extremity of the left middle turbinated body, extending beyond the orifice of the Eustachian tube. It was removed by curved forceps passed behind the palate, under rhinoscopic guidance, and without cocainization. The bulk of the mass was about that of a bean.

7. *Pleuro-pneumonia; lung in state of gray hepatization; congested kidneys; purpuric eruption of intestinal mucous membrane.*

Exhibited by Dr. D. J. M. Miller.

James N., æt. 50 years; single; white; laborer; of intemperate habits; admitted to the Episcopal Hospital on April 11, 1888. Had been perfectly well until four days before admission, when slight chills occurred, followed by stitch-like pains over right chest, cough, headache, and diarrhœa. Examination revealed an extensive pleurisy on the right side. On the 13th great increase of pulse, respiration, and temperature was noted; the cough had become hacking, the sputa tenacious and rusty, and faint bronchial breathing, increased voice resonance and dulness on percussion were observed for the first time over the root of the right lung. The diarrhœa had increased; there was much tympanites. The pneumonia rapidly spread, so that in four days it had involved the whole of the right lung, except its lower fourth. The temperature then began to fall, and in three days was normal. The condition of the lung remained unchanged during the next eight days; the diarrhœa continued. About this time facial erysipelas made its appearance, and remained until the 16th of May, when a profuse purpuric eruption appeared on the face, spreading in a couple of days to the arms, loins, and thighs. It was accompanied by hemorrhages from the gums, nose, bowels, and kidneys. He died on the 20th from exhaustion.

Autopsy, eight hours after death.—The right lung was found everywhere closely adherent to its pleura, so that the two had to be removed together. The lung itself was consolidated, and airless, except at the base of the lower lobe, and appeared to be in a state of gray hepatization. The liver was enlarged, and filled with dark, venous blood. The kidneys were enlarged, weighing ten ounces, and intensely congested; the calices filled with clotted blood. The heart seemed normal. The spleen congested. The mucous membrane of the large intestine was dotted with numerous purpuric spots, similar to those seen upon the skin. The same condition was observed throughout the smaller gut. The stomach was unchanged. For the notes of this case I am indebted to Dr. W. T. Van Pelt, the resident physician. *May* 24, 1888.

8. *Acute phthisis; erosion of a large branch of the pulmonary artery; sudden fatal hæmoptysis.*

Exhibited by Dr. W. Osler.

A colored man, æt. 33 years, had been in the Philadelphia Hospital some weeks with pulmonary tuberculosis, a cavity of the left apex, and extensive involvement of the upper part of both lungs. The patient was up and about. Suddenly, in the evening, without any premonition, he began to cough up blood in large mouthfuls. In the space of a few minutes he fell back dead. The *autopsy* showed extensive tubercular lesions, cavities at both apices, small cavities at the upper part of the lower lobes, very many small foci of softening. The bronchi and a majority of the cavities contained small blood-clots. A systematic dissection was made of the bronchi and pulmonary artery, all the branches of which, passing to the lobes of the right lung, and to the upper lobe of the left lung, were slit open without finding the source of hemorrhage. In the lower left lobe, close to the root, a small cavity the size of a walnut was opened, which contained clots, and which had undermined a large branch of the pulmonary artery. Projecting from its lumen into the cavity was a small sac, 3 by 3 mm., at the apex of which was a linear rent, communicating with the cavity. The cavity opened directly into a large bronchus. Dr. Osler remarked that this was a very common condition in the fatal hemorrhage of chronic phthisis. The lesion is either aneurismal dilatation or erosion of a branch of the pulmonary artery in the wall of the cavity. In this case a large vessel in direct communication with a bronchus of at least the third dimension explained the rapidly fatal character of the hemorrhage.

October 25, 1888.

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9. *Colitis; gangrenous infarct of lung; pneumothorax.*

Exhibited by Dr. W. Osler.

The specimens were removed from the body of an old woman who was admitted from the almshouse to the medical wards of the Philadelphia Hospital with diarrhœa. She was in a condition of extreme debility, and could not give any satisfactory account of her illness. She

was short of breath, and had a cough, with much expectoration. Death occurred about forty-eight hours after admission.

In the left pleura there was air and a moderate amount of sero-purulent effusion. The heart in fully one-half of its breadth lay to the left of the median line, and the relative position of the apex and base was maintained without any twisting of the organ upon itself. On the anterior margin of the upper lobe of the left lung was a V-shaped gangrenous infarct, directly over the pericardium, and beneath the third and fourth intercostal spaces. Pressure upon the infarct caused air to bubble from beneath the attached edge. On lifting it a small slit-like perforation was seen. The lower lobe of the lung was collapsed and airless; the apex and anterior third of the upper lobe contained air. The pleura over the infarct was grayish in color, covered with fibrous lymph, and the lung tissue in its immediate neighborhood, for a distance of one and one-half inches, was solidified.

Section of the infarct showed it to be in a condition of gangrene. The tissue was shreddy and dark in color, and infiltrated with a sanious fluid. The odor was extremely putrid. On tracing the pulmonary artery passing to this part a thrombus was found in the branch entering the infarct. The plugged artery was firm and hard, and for nearly an inch of its course was surrounded by a disintegrated, dark-looking tissue. There were no other areas of infarction in either lung. A moderate amount of lymph covered the costal and visceral layers of the pleura of the lower lobe. The heart was small, and the valves a little atheromatous. There were no thrombi in the right chambers. The kidneys and spleen showed senile changes. The mucous membrane of the colon was swollen, in places deeply congested, and there were many small follicular ulcers. In parts of the descending colon the swelling of the mucosa was unusually great.

Dr. Osler remarked that pneumothorax from this cause was not so common as from the perforation of a tuberculous abscess. Last year he had shown a specimen of the hemorrhagic form which had ruptured, and caused a fatal termination in a case of heart disease.

November 8, 1888.

10. *Empyema due to caries of the dorsal vertebræ.*

Exhibited by Dr. J. C. Wilson.

Mary T., negress; æt. 34 years; unmarried; housemaid; admitted to the Women's Medical Ward of the Philadelphia Hospital, on November 2, 1888; family history unknown; patient has had two living children, and several miscarriages; has suffered from kyphosis in dorsal region since childhood; history of cough, expectoration, and failing health for several months. Patient was treated in the nervous wards of the Philadelphia Hospital eighteen months ago for incomplete paraplegia, from which she wholly recovered; in September she caught cold and began to suffer from fever, attended with increased cough and rapid failure of strength.

On admission heart-sounds feeble, action rapid and irregular; pulse 140; no cardiac murmurs; respiration shallow, rapid, superior costal, 60 to 70 per minute; hacking cough, with expectoration of purulent matter at intervals varying from several hours to a day or two; right chest resonant over upper lobe, with feeble respiratory murmur, dulness of base anteriorly and posteriorly, with absence of respiratory murmur, etc.; left chest resonant throughout; respiratory murmur feeble, well-marked friction sounds at base posteriorly; urine albuminous, and contains hyaline, epithelial, and granular casts; tongue clean and pale; moderate appetite; no vomiting; daily movement of the bowels; moderate febrile movement of irregular remittent type; evening temperature not exceeding 102° F. Four days after admission the dyspnœa suddenly increased; respiration 80 per minute; pulse 150; great oppression; orthopnœa. Dr. Sharpless, resident physician, withdrew by aspiration from the right chest 23 fluidounces of thick, yellowish pus, which gave great relief for the time, but the symptoms recurring, it became necessary to repeat the operation six times in the following ten days. The amount of pus withdrawn varied from 6 to 12 fluidounces. Death occurred suddenly on November 19, while the patient was in the act of drinking water.

Post-mortem examination.—Moderate emaciation; protuberent abdomen; antero-posterior curvature of dorsal spine.

Heart: Large; all cavities contained fluid blood; no valvular disease; edges of tricuspid leaflets slightly thickened.

Lungs: Old pleural adhesions at apex of left lung; more recent adhesions at base on the right side; thickened pleura, with pus cavity,

presented toward antero-lateral surface of chest. Right lung, upper lobe crepitant, lower and middle lobes compressed and airless. Left lung crepitant throughout; no tubercle nodules or infiltration in either lung. The pleura forming the walls of the pus cavity thickly studded with tubercle. This cavity contained several ounces of thick, creamy pus, and some air, forming a pyo-pneumothorax. Careful examination failed to reveal any connection with a bronchus. Mediastinal glands enlarged, deeply pigmented, not caseous; the lateral borders of the fifth and sixth dorsal vertebræ roughened and carious.

Liver enlarged and fatty.

Kidneys large, pale; presented the microscopic appearances of catarrhal nephritis.

Spleen normal.

Intestines showed no tubercular ulceration, and mesenteric glands were not enlarged. *November* 22, 1888.

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11. *Pseudo-membranous laryngitis produced by inhalation of flames.*

Exhibited by Dr. H. F. Formad.

The specimen was removed from the body of an insane man, who had kindled a fire in his cell, and standing immediately over it had inhaled the flames. Death resulted forty-four hours after this ill-considered act. Preceding death there had been prominent laryngeal symptoms, croupy cough, stridor, dyspnœa. The larynx and trachea were found lined with a membrane as typical as any that could be found in a case of true croup. This condition proves conclusively to my mind that pseudo-membrane is not necessarily due to any specific micrococci, but may occur as a result of a direct injury to the mucous membrane. And if to a direct injury, why not to a simple exposure to cold? So that we may say with truth there are two kinds of croup, one arising from cold without the intervention of any specific cause—true croup; the other the result of a diphtheritic process—diphtheritic croup. In addition to this lesion in the respiratory tract there was in the upper part of the duodenum an intense catarrhal inflammation, with scattered points of ecchymosis; the remainder of the intestinal mucous membrane was devoid of lesion. This condition of the duodenum is of extreme interest, as showing the mode of formation of a duodenal ulcer following a burn through an initial catarrhal inflammation succeeded by ecchymosis. Had

this poor misguided creature lived a few days longer, nothing is more certain than that I should have found, instead of inflammation, an actual ulceration. *December* 13, 1888.

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12. *Pyo-pneumothorax.*

Exhibited by Dr. J. K. Mitchell.

O. W., æt. 26 years; native-born; white; cigar-packer; was admitted to the University Hospital October 19, 1888. His father, a German, died of phthisis. His mother and four brothers are living, and well.

His personal history was good until an attack of typhoid in January, 1888, since which time he had never felt thoroughly well. The typhoid was preceded by two months of chills, almost every other day, followed by slight sweating.

For about two weeks before admission he suffered with some shortness of breath, otherwise he could give no details which would fix the onset of his illness.

Examination.—Mensuration: Right chest, 15½ inches; left, 15⅝. Expansion: Right, 1 inch; left, none. The cardiac shock is felt beneath and to the right of the sternum. On percussion, in a semi-recumbent position, there is defective resonance below the left clavicle, and hyper-resonance from the left subclavian region to the lower margin of fifth rib. Right side clear. Erect, dulness begins at lower edge of third rib, on left side; below this there is absolute flatness. Posteriorly the right side gives a clear note; the left scapular region is somewhat resonant; dulness noted over the rest of the lung.

Upon auscultation crackling râles are heard on the right side down to the third rib, with loud, harsh respiration. On the left, high-pitched tubular respiration is heard in the infra-clavicular region. The heart-sounds are distinctly transmitted.

On the left side a clear metallic tinkle, at the end of inspiration, more distinct on taking a deep breath, is heard as low as the fourth rib. Posteriorly, the left apex presents numerous râles, mucous, piping, and sibilant in character, extending into the interscapular area. The breath-sound is tubular and rough, and below the apex is quite clearly heard, with no râles. At the level of the fifth dorsal vertebra there is a distinct metallic tinkle, and below this silence. On percussion with two coins the "bell sound" is well developed, and a very pronounced succussion

splash may be had by shaking the patient. An irregular fever, ranging up to 103°, persisted during his stay in the hospital. During the first six weeks his condition changed very little, but in the first week of December he became suddenly delirious, and with brief intervals remained so until his discharge, by request, on December 23. I attended him at his home, noting but little alteration except the gradual disappearance from the left chest of all trace of a respiratory murmur and a tubular inspiratory sound in the second interspace, until January 8th, when he died.

Dr. Osler kindly assisted me at an *autopsy* forty hours after death, Drs. Daland and Wood being also present, when the following notes were made:

Percussion gives a typical tympanitic note in the infra-clavicular and mammary regions on the left side.

On removing the intercostal tissues from the second, third, and fourth interspaces, the left border of the heart is seen to lie just inside the left para-sternal line. The interspaces are unusually wide. The apex of the heart corresponds to the fifth interspace, and lies 1½ inches to the left of the mid-sternal line. The extreme right border of the heart is in the third right interspace, 2¼ inches from the mid-sternal line, and in the second interspace the auricular margin lies 2⅛ inches from the same line. The left pleural cavity has a large air space above, while about a quart of laudable pus occupies the lower part. The remains of the compressed and carnified lung are pushed up into contact with the side and posterior surface of the pericardium, and adhere behind to the costal pleura. The general position of the heart is not much altered. The right auricle seems unusually elongated, the appendix is vertical instead of transverse, and the right ventricle seems to compress the lower part of the auricle and the termination of the inferior vena cava. A number of ulcerative openings through the lung and pleural surfaces appear at the left apex. The costal surfaces are covered with a deposit of pus, easily removed, showing only the bare surfaces of the intercostal muscles, with no trace of the costal pleura. The diaphragmatic pleura is also much eroded on the left side. On the right the pleuræ is universally adherent.

The right *lung* is large, heavy, has a small localized apex-cavity, extensive old tubercular deposits, and numerous tubercles in the pleura. The whole of the lower lobe presents recent eruptions of tubercles.

The *stomach* is much dilated, reaching to within 2 inches of the symphysis. The peritoneum and intestines show no signs of tubercular deposit.

I ought to add that repeated examinations of the sputum has failed to demonstrate the bacilli of tubercle. *January* 24, 1889.

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13. *The larynx of an infant dead of laryngismus stridulus.*

Exhibited by Dr. D. D. Stewart.

The infant, æt. 5 months, from which this specimen was taken, was admitted to St. Christopher's Hospital the day preceding its death to be treated for convulsions and laryngismus stridulus, present since birth. It appeared well-nourished, and was not rhachitic. There was no cough, and a physical examination of the chest failed to elicit any signs of enlargement of the bronchial glands. The laryngismus was quite constant, though more or less distinctly aggravated by excitement. While the nurse was dressing the infant to take it to my office (a short distance from the hospital) for a laryngeal examination, a severe convulsion occurred, which ended fatally.

Post-mortem: The epiglottis was found somewhat more curved in its long axis than normal, though apparently not to a sufficient extent to produce complete approximation of the ary-epiglottidean folds, and thus originate the paroxysmal inspiratory dyspnœa, as in the case of Dr. Lees (*Path. Trans. of London*, 1882). There was no atrophy of the posterior crico-arytenoid muscles. I am unable to satisfactorily account for the laryngeal spasm. *February* 14, 1889.

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14. *Bullet encysted in the lung.*

Exhibited by Dr. H. F. Formad.

The man from whom this bullet was removed had been wounded in the chest at the battle of Gettysburg. After a long and serious illness following the wound he recovered perfectly, and continued well, as far as the lung was concerned, complaining only occasionally of slight pains in the chest, which caused him merely a temporary inconvenience. He died of conditions entirely unconnected with the bullet wound. At the *autopsy* the bullet, flattened and twisted entirely out of its ordinary shape, was found in the extreme base of the lung, resting upon the diaphragm. It was completely encysted in a dense fibrous capsule. About

it the lung was apparently perfectly normal, giving no evidence of any recent or old inflammatory change. The scar caused by the entrance of the bullet through the chest-wall was just above the nipple, and it seems probable to me that the bullet reached its present position by slowly settling down through the lung until arrested in its course by the diaphragm. *April* 11, 1889.

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15. *A fatal case of Ludwig's disease, pyæmic pneumonia.*

Exhibited by Dr. A. A. Eshner.

W. P., æt. 39 years, a native of Denmark, was admitted to the ward of Dr. Steinbach, at the Philadelphia Hospital, on June 5, 1889, with moderate swelling in the region of the left angle of the jaw, headache, difficulty in separating the jaws, and dysphagia. The swelling was said to have begun four days before. The breath was fetid. Examination of the buccal cavity revealed the presence of sloughing tissues, with a sense of fluctuation. On the day following admission the temperature was found to be 103.6°. The swelling progressively increased, extending upward over the face, downward over the neck, and to the opposite side, and the other symptoms likewise became more aggravated, while the temperature fluctuated between 101° in the morning and 103° in the evening, and the respiratory movements increased in frequency from 24 to 44 per minute, the alteration in the frequency of the pulse being insignificant. On June 8 an incision was made into the outer surface of the gum of the lower jaw, on the left side, below the molar teeth, and the bone was found denuded of its periosteum, the teeth carious. On June 10 the patient was anæsthetized, and the diseased structures removed, the bone proper proving to be but little involved. An incision was made in the median line, over the symphysis of the jaw, and another at right angles to this along the lower border of the left half of the body of the bone. There was moderate sloughing of the soft structures, and pus and serum welled out of the cut textures. The bone was found denuded of its periosteum. The two posterior molar teeth, one of which contained an abscess, were removed, the soft structures curetted, and the wound treated in the usual manner. It is proper to state here that chloroform was used as the anæsthetic, less than four ounces having been used, and the patient scarcely at any time being under the complete influence of the drug, much being lost by evaporation from the towel. The patient readily returned to consciousness, but became delirious,

tearing out the drainage-tube from his mouth, and endeavoring to remove the dressings, so that forcible restraint became necessary. Following the operation the temperature began to rise, reaching 105° at 2 and at 4 o'clock, 106.2° at 6, 107.4° at 8. Death occurred at 8.20 P.M., some eight hours after the operation, which occupied, perhaps, an hour. The temperature then taken in the rectum was 109°.

The *autopsy* was made fifteen hours after death, under the direction of Dr. Formad. The *right lung*, at its upper lobe, anteriorly, was found adherent to the thoracic parietes. The lungs were highly congested, and œdematous. In various parts of the periphery of both lungs, but principally in the middle and lower lobes on the left, were found numerous hemorrhagic infarcts, varying in size from a pea to a hazelnut, some of which displayed distinct purulent softening, indicative of their pyæmic origin.

The *pericardium* contained two or three ounces of clear, serous fluid.

The *heart* was normal in size, and displayed no lesions, save a few patches of chronic endocarditis at the margins of the mitral leaflets, and upon the aortic valves. All the cavities contained large, firm, chicken-fat clots, which extended for some distance into the large vessels.

The *liver* was somewhat enlarged, and in a condition of fatty infiltration.

The *spleen* was enlarged, and softened from septic change.

The *kidneys* were in a condition of chronic parenchymatous nephritis, in the second stage.

The *peritoneum* was normal. There was some swelling of the mesenteric glands, one of which was as large as a walnut, and had undergone calcareous change. *June* 13, 1889.

V. THE GENITO-URINARY APPARATUS.

a. KIDNEYS, BLADDER, ETC.

1. *Tuberculosis of kidneys.*

Exhibited by Dr. J. H. C. Simes.

Patrick H. was admitted to the surgical ward of the Episcopal Hospital on the afternoon of November 2, 1887, suffering with retention of urine. The resident, Dr. H. C. Deaver, succeeded in passing into the bladder a No. 4 (French scale) English catheter, through which the urine drained. This was left in the urethra twenty-four hours, when a No. 8 was introduced, and remained in position twenty-four hours. He was now able to pass his water in a fair stream, but small. On November 6th I introduced into the bladder Nos. 14, 15, and 17 steel sounds. November 9th Nos. 15, 17, and 19 steel sounds were passed without any difficulty. This same afternoon, toward evening, the patient had a chill, and at night he had retention of urine. A catheter was introduced into the bladder, and bloody urine drawn off. The next morning, November 10th, there was considerable distention of the bladder, evinced by percussion, and also pain over the hypogastric region, with inability to pass urine. A catheter passed without difficulty into the bladder, but there was no flow of urine. Upon withdrawing the instrument its openings were found occluded by blood-clots. A very weak solution of bicarbonate of soda was injected into the bladder, and a disintegration of the clot took place. This relieved the over-distended bladder, but there was no decrease in the pain, nor was he able to empty his bladder, which necessitated the passing of a catheter every three or four hours. The treatment consisted in the employment of hæmostatics, both by the mouth and injected into the bladder. Quinine in large doses was also tried. Nothing had any influence in controlling the hæmaturia. The patient gradually became exhausted, and died on the morning of November 12th.

Post-mortem, made by Dr. Deaver, two hours after death.—The thoracic cavity showed no pathological lesions, except that both lungs presented

some points of inflammatory induration, and an extreme condition of anthracosis. The man had been employed on the coal wharves of the Reading Railroad. In the abdominal cavity there were no morbid changes in any of the viscera, with the exception of the kidneys and bladder. The left kidney was about twice the normal size, and the entire organ infiltrated with tubercular formations. The right kidney was somewhat contracted in size, and presented a few foci of tubercular growths. Both ureters were very much thickened, and dilated to three times the normal size. About two inches from the junction of the left ureter with the bladder there was found a focus of ulceration, which was evidently a seat of hemorrhage, the surface being covered by recently clotted blood. The bladder was found to have its walls very much thickened, measuring half an inch, its cavity much contracted, and the mucous lining eroded and ulcerated. The urethra showed a stricture at the bulbous region, which was evidently the seat of obstruction to the introduction of instruments, previous to its dilatation. An interesting feature of this case, aside from the morbid condition found at the post-mortem, was to determine the cause of the hæmaturia during life. The possibility of the hemorrhage being occasioned by a false passage, made in passing the instruments, presented itself first to my mind, but a careful examination and consideration of the symptoms lead me to exclude this as the cause of the bleeding. An indirect history of malaria pointed to this affection as being a possible cause, for which quinine was given; but later the malaria was also excluded, and I finally felt satisfied that there existed some lesion of the bladder, the nature of which I was not able to determine, thinking, however, it might possibly be a growth within the organ. This opinion was further rendered more plausible by the circumstance that he had had for the past year, previous to his attack of retention, incontinence of urine. Tuberculosis, however, was not suspected. The question of an exploratory operation by perineal section was thought of, but due consideration lead me to believe it was not justifiable, and, as the post-mortem showed, it would have been useless.

Report of the committee on morbid growths:—"Your committee reports, that the kidney presented by Dr. Simes is found to be the seat of extensive amyloid disease, involving especially the arteries in the pyramids. Many of the glomeruli are transformed into fibroid balls, in which small deposits of amyloid material can be demonstrated. There is marked fatty degeneration of the epithelium, the convoluted tubules being almost completely denuded.

"The specimen examined by the committee showed everywhere

inflammatory process characterized by abscess formation and infiltration of the interstitial tissues by small cells. These small abscesses show a tendency to become confluent, especially in the cortex, where the process is more marked. In many places, in the centres of the abscesses and in the tubules and in the glomeruli, zoöglœa of micrococci may be seen, often arranged in chains. No tubercle bacilli can be found.

"The ureter presented macroscopically small defects of tissue, with hyperæmia, and microscopically an infiltrate below the denuded mucous membrane, with extensive hemorrhage. No bacilli could be found in sections, but a few cocci on the surface."

December 8, 1887.

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2. *Specimens of hydronephrosis.*

Exhibited by Dr. F. P. Henry.

The patient from whom these specimens were obtained was a man æt. 39 years, who had suffered for many years with incontinence of urine, and was obliged to wear an apparatus to collect it as it flowed, drop by drop, from the meatus. His trouble dated from an injury received about twenty years ago, at which time he was employed in a hardware establishment in New York. The injury was caused by a box filled with metal goods falling upon his spine. I have been unable to ascertain the size of the box, its weight, or the distance from which it fell. Suffice it to say, that the injury was so severe that he was taken to a hospital, but that there was no paralysis is proved by the fact that on the following day he left the hospital and came to his parents in Philadelphia. Some time after, his relatives cannot give any definite idea of how long, he was sounded for stone, with a negative result. There is no doubt that he had suffered from incontinence from a period shortly after the receipt of the injury above mentioned. At various times, also, he has had attacks of hæmaturia. I had never been consulted with reference to his bladder trouble until February 25th, when I was told that he had passed no urine for nearly twenty-four hours. After considerable difficulty occasioned by the spasmodic action of an irritable urethra, I passed a catheter and drew off two or three drachms of bloody urine. The only complaint at that time was of pain in the region of the left kidney. I saw the patient the next morning, and learned that he had a sleepless night, for the most part, and had passed little or no urine. The pain in the left loin, which had been absent during the

night, had returned. I again passed the catheter, and brought away a little blood. This was not due to the operation, which was performed with the greatest care. Not feeling satisfied with my efforts to relieve the patient, I summoned Dr. Nancrede in consultation in the afternoon of February 26th. He passed a catheter, and no urine following, it was withdrawn with a clot an inch long attached. After this a few drachms of bloody urine came away. It was obvious from the sensation communicated through the catheter that the bladder was greatly contracted. On the evening of the 27th the pulse, which had been from the first day of my visit above 100, reached 120, and there was some twitching of the upper extremities. The patient was quite rational, but his expression was wild. The urine continued bloody, and came away in very small amount. He died in a slight convulsion on the morning of the 27th.

The autopsy was made the same afternoon. There were signs of slight recent peritonitis over the coils of intestine occupying the lower portions of the abdomen. The left kidney was about three times the normal size, and consisted of a sac containing a sanguinolent fluid. The walls of this sac are, as you see, exceedingly thin, and present no appearance of kidney tissue. No calculi were found in any portion of kidney or ureter. The latter was dilated so as to resemble precisely a coil of intestine. The right kidney presented externally the same appearance as the left, that is, it was lobulated and fluctuating, but smaller. On opening it, it was found to contain pus, both liquid and in a concrete mortar-like condition. The lumen of the right ureter is occluded. Owing to the circumstances under which the autopsy was made it was difficult to remove the bladder entire. The portion shown, which contains the orifices of the ureters, shows that it was greatly contracted.

In 1879 I presented a similar specimen to this society, and argued that even in the absence of obstruction to the outflow of urine hydronephrosis might occur. To quote from the report of that case: "In uncomplicated cystitis the bladder is never distended; its capacity is diminished by the thickening of its walls and the state of contraction in which it is habitually maintained. This contraction, which is great, becomes extreme so soon as a few drachms of urine have collected, and, as a consequence of the disordered function, the ureter and pelvis of the kidney have to serve vicariously as receptacles for the urine. In health the bladder is passive except during micturition, and offers no resistance to the function of the ureter; in cystitis the function of the ureter is

interfered with, and the urine reaches the latter largely by hydrostatic pressure."

The inspissation of the pus in the right kidney was probably consecutive to occlusion of the ureter.

I have just received the following note from Dr. Nancrede, to whom I wrote inquiring whether, in his opinion, there was any stricture in this case:

"There was no organic narrowing, but a distinct spasm of the whole canal." *March* 8, 1888.

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3. *Some points in urinary pathology.*

Read by Dr. Morris Longstreth.

The materials which I have the pleasure of offering to your consideration this evening cannot pretend to the dignity of a complete discussion, of even any one subject, in the wide field of pathology and work which the title of the evening's discourse has announced to you.

When our President, but so shortly ago, requested me to occupy the speaker's position before you, I found myself barren of matter in a state ready to be offered here to-night, and the time was too brief to prepare a question with new work in a befitting manner, worthy of presentation to you. But, I am willing and glad to do my duty as a member of the society, and to assume the orator's mantle—the mantle which, I think, should be on all of your shoulders, and which would adorn so many of you.

What I propose for you to-night, in the absence of any new discussion, is to pull out a leaf here and there from my note-book of urine examinations, and to offer the facts therein recorded, with such comment as the matter or cases seem to need. These leaves can furnish necessarily only a series of disconnected observations, and it is my further purpose, out of the mass of facts, not to weave together the things which go to prove the rules or regular things of urine pathology and urine examinations, for these things you already know. I would much rather try to present to you the exceptions to the rules. Exceptions do not prove the rule, as the old Latin proverb hath it, but in medicine, as in other questions of nature, the exceptions are more interesting, more striking than the routine matters, and withal often more valuable and instructive in solving the problems which hourly and daily come before us for examination.

I do not intend to present to you any exceptions to the well-founded rules of urine observations, as they have chanced to occur to me, merely as curiosities—let that be far from me—but only so far as they may be of service as illustrations of the rules, and also to widen our knowledge—I was going to say our ignorance—of kidney function in diseased conditions.

I find that my records of urine examinations, in the first place, fall naturally into two great groups or classes: 1st. Those cases, and they form the larger number, in which the examination was carried on, not for the purpose of distinguishing Bright's disease or any essential kidney malady, but for the purpose of determining the aspects of general nutrition and assimilation. 2d. The groups of cases including Bright's disease and the numerous other conditions of organic alterations to which the kidneys are liable. Besides these two classes of cases there are, I find, a mass of observations, which I value not less highly, on normal urine, carried on for the purpose of making the clinical records of the cases complete, in which there was no suspicion of renal disease, and in many instances no other trouble whatsoever was suspected. These observations of normal urine I prize thus so highly because they are normal cases, and because, while making them, I did not know the urine was normal. Much the larger number of them were done on my part in ignorance of the clinical history of the cases, done for members of the profession and for filthy lucre, carried on with the expectation and the endeavor of finding some abnormal condition. In most of these normal cases, subsequent to the examination, I obtained a history which showed the kidneys not to be at fault, and in not a few of them the autopsy proved the kidneys were not the seat of disease. Too often it happens that one does not have the courage and persistence to search normal urine in due measure to know it on sight. Without the stimulus, and under good conditions for straining one's acumen, unbiased by the knowledge of the patient, this work is too constantly left undone. Yet, how is one to know urine if one is always looking only at the secretion of Bright's disease, or of a nervous dyspeptic? One has only the resource left of following the *rules and regulations*, and rules fail to guide one aright in any natural study.

Turning to that group of cases in which we are not looking for essential or organic kidney disease, but for mischief or perverted function in other parts of the body—those nervous and dyspeptic cases with perturbed and perverted nutrition, and many eccentric phenomena, causing great discomforts, and as shown by several of my records, permanent bodily and mental wreck. Of these instances the most striking I find

in my records are *calcium oxalate* cases. The field of discussion of oxaluria, as the condition has been denominated, is a very wide one, and is, moreover, it seems, one but very imperfectly comprehended in its nature and characteristics. I shall endeavor on the present occasion not to extend the field of discussion concerning the chemical origin of oxalic acid; but, on the other hand, limit myself to describing some of our means for its recognition, and only incidentally speak of the states with which it is connected. Oxaluria, as Golding Bird spoke of it, is a very important condition, and, if the term is properly restricted, is one which we can make use of to-day with great advantage. But it requires close pruning, as it is used to-day, in order to be valuable. In the first place, oxaluria must be distinctly understood *not* to be the mere finding of crystals of calcium oxalate in the urine. There are a number of states of urine in which oxalates are present so very unlike that they offer no points of similarity except the oxalate crystal, and in some of them the crystals appear so unlike that, from this feature alone, a differential diagnosis can often be made between the conditions. A chief feature for the diagnosis of this restricted oxaluria—the true oxaluria—is the regular and constant occurrence of the crystals, and especially in the morning secretion, and that coming after a full meal. Oxalates of other forms, and from other conditions, are generally intermittent, coming by spells, irregularly, of varying duration, often less in the morning, but especially not influenced in coming by food-taking. Again, the appearance of the urine itself in true oxaluria, after it has stood in a proper precipitating glass, is alone almost diagnostic of the condition, or at least very characteristic: an appearance not easy of description, one that must be learned by frequent repetitions of the sight. A urine which is pale and clear when fresh, but after standing forms a light cloud or clouds—and these clouds seem to form successively—which do not tend to settle deeply even after prolonged rest, and, if I add a low specific gravity, will, eight times out of ten, be the secretion of an oxaluric patient. These clouds, which hang suspended, will sometimes be seen to glitter when held in a proper light. The typical specimens show a striking resistance to putrefactive changes, and continue unclouded even after the microscope reveals abundant bacterial life.

The microscopic features of the urine and of the oxalate crystals themselves are, to my thinking, thoroughly diagnostic, and conclusive of the true oxaluria as distinguished from other urines which may contain oxalate crystals. One sees in the field of view, when the drop for examination is selected from a cloud of the precipitate, a meshwork of strings of mucus, with usually rather abundant numbers of mucous

corpuscles in or around the mucus strings, and then oxalate crystals varying in number with the individual case in hand, or with the period of day when the urine was passed. The mucus is regularly and continuously present—that does not seem to vary very much as long as the oxaluric state lasts.

But it is to the appearance of the oxalate crystals that I look to make a diagnosis of oxaluria—the true oxaluria. The crystals are in fresh samples of urine, that is, within six hours, always of the octahedral form. I have no instance recorded of dumb-bells in true oxaluria. Moreover, I have always found these crystalline forms to be, when early examined, of small size, and in distinct pieces, not conglomerate masses. The smallness and evenness of size of these oxaluric crystals is a striking feature of them. I have never seen them occur in gigantic crystalline masses, such as are frequently seen in other urines not oxaluric. They often come of most minute size, looking at first glance like a trifling collection of amorphous urates, but a little washing, a little careful handling, and sufficient magnification—700 or 800 diameters is sometimes necessary—will show out their true octahedral form. I have been able to show these crystals readily by such enlargement, which otherwise could have been easily passed over undiscovered—whole armies of them can thus be disclosed to view, where, under lower power, only specks were seen—but I do not think the high power is essential after one has recognized the speck as an oxalate, and become accustomed to its deceptive appearance. The oxalates of oxaluria, I have already said, do not come as dumb-bells; neither have I found them joining on to each other, making irregular crystalline masses, or those so-called microscopic calculi, as oxalate of lime crystals so frequently do in genuine calculous disease, in some purulent urines, and in urine secreted after the ingestion of vegetables and fruits containing the material as an ingredient.

As to the other instances, not oxaluria, in which oxalate of lime crystals occur, I have been able to determine no gross appearances characteristic of the urine; of the microscopic appearances, they are very irregular and inconstant. Of some of these I hope to speak later on in connection with renal calculus. There is one word of caution worth the while to utter before leaving oxalate crystals, and that is the enormous crystals may be mistaken, when massed together or broken, for fragments of triple phosphates—I have known this error to occur—and the dumb-bells will resemble sometimes the radiating formations of phosphate of lime.

Of phosphoric acid, as represented by phosphate crystals, I have little

or nothing to mention that is of interest and of use. This ingredient has always seemed to me more regularly constant, at least proportionately, in amount than any of the other normal matters we customarily search for by analysis. I cannot forbear to mention the value I have derived from the recognition of the presence of ammonio-magnesian or triple phosphates, in determining or confirming the existence of bladder troubles in otherwise doubtful cases. I have made several records of purulent or muco-purulent urine of alkaline or neutral reaction, but without triple phosphates, in which the disease was of the kidney, and not bladder, as shown conclusively by the subsequent clinical phenomena. I have recorded instances of bladder disease with acid urine, in which the origin of the abundant mucus was explained, and a correct diagnosis arrived at, through the discovery of constantly coming phosphate crystals.

On diabetic or saccharine urine I have a number of curious and valuable records. But let me first speak of the methods and the appearance or results of the tests. Personally I prefer, and always primarily, the copper test, which goes by the name of Fehling's. Everyone must have been bothered by finding this test-solution out of order in some way at the moment when in the greatest hurry for examination, and just when there was the least time for replacing it. I have lately been using a modification of the original, which has stood well the test of time both for accuracy and keeping properties. It is not of my invention, but I am not able to give the credit to the possessor of the merit, for I have forgotten where I obtained it. The following is the formula for its preparation: Crystallized sulphate of copper, 17 grammes, dissolved in 100 c.c. of water; pure mannite, 7½ grammes, dissolved in 50 c.c. of water. Add the two solutions to a solution of caustic soda, sp. gr. 1145, 240 c.c. Add then sufficient water to make ½ litre. It is to be used in the same manner, and with the same precautions, as Fehling's test solution, and the bottom contents of the bottle will be found as satisfactory as the freshly made solution. Here is a sample which has been made a considerable time, and is perfect still. If any doubt arises as to the presence of sugar, resort should be had to the results of fermentation. Let me show you this apparatus. It was devised by Einhorn, and with easily followed directions.

In the finding of sugar in urine there is ordinarily nothing more simple, but I think the routine testing for it is too frequently omitted, dependence being placed on the clinical history or the specific gravity to presume its absence. But sugar is tricky sometimes, and whilst no one would omit the test for a urine above 1030, yet sugar occurs in

urine of low specific weight. I have two instances to record of saccharine urine at or below 1015. The lowest was in a urine of Dr. William Hunt's practice, 1012. In a case of my own it was found in a urine with a specific gravity of 1015. In my own case the patient was not fairly to be called a diabetic, since there were no accompanying symptoms which go to make up the usual picture of this disease.

On the other hand, sugar is attributed to a urine which in reality contains none of it, through mistakes or misapprehension of the results of the chemical tests. Most of these errors come in the way of misjudging the point of reduction of the copper to the sub-oxide. Many are content to accept as evidence of sugar a discharge of the blue color of the test solution, and with it the production of dirty yellow precipitate, without being assured of the appearance of the reddish sub-oxide. The dirty yellow often enough comes along with the reddish oxide, but the red is essential for certainty. I have a group of records illustrating this error. It is seven years ago, so my note-book says, that a medical friend forwarded in quick succession six samples of urine, with the request for an examination. We two did not meet until the six examinations were completed. His first question was, Did you not find sugar? No, I replied, as my reports had already shown him. Well, he said, that is strange. I have been thinking for a year or more past that I had an unusual number of diabetic patients—more than in all my life before. He had sent the same urines as had been forwarded to me to a chemist, with the regularly recurring report that they were saccharine. The explanation was ready at my hand. I had produced a dirty yellow by boiling the urine with the Fehling's solution, and in one of the specimens there seemed doubtfully to be a trifle of red precipitate. But, the whole reaction for sugar was so uncertain that the crucial trial by fermentation was demanded to determine the fact. These urines all failed to ferment on the addition of yeast, although they were able in a partial manner to reduce the copper. There are not a few urines which are able to act in this manner, but from the presence of other ingredients than sugar in them; they are almost invariably urines with a high specific gravity, and are, therefore, from this feature of them, in a sense misleading. There is no excuse for this negligence in testing. The fermentation process does not require a special apparatus. We, all of us, let sample bottles of urine stand around a longer time than it takes to make this examination, and the test demands so little more labor. If one will drop a small fragment of yeast cake—and, in these days of Fleishmann, yeast is in every household—and cork the bottle lightly, it needs no watching, it is self-recording. All one has to do further is, after

the bottle remains in a warm place a few hours, to hunt for the cork, provided the specimen contains sugar. The carbonic acid and the cork together furnish an ample register of the condition, and it is not necessary to exercise one's judgment about a change of color. Furthermore, the reduction of the specific gravity is one of the most certain relative measurements of the amount of sugar contained in these urines, although not a very convenient method compared with the volumetric copper expedient.

Turning now to the second group—those urines in which are found evidences of organic alterations of the kidney and the urine conducting apparatus—the thing which first of all strikes our attention as a morbid ingredient in these urines is albumin, at least in the vast majority of the cases. We encounter this material first in the routine examination, and by its presence our search is keenly sharpened for further evidences of organic alterations. In fact, the mere appearance of albumin keeps in our minds a constantly lingering suspicion that all is not right with the kidney which furnishes it, at least until a clear explanation of the origin of the occurrence. Even the disappearance of the albumin does not remove the suspicion without the clear explanation.

Yet, just how valuable is the appearance of albumin, with or without other features, in convincing us of the reality of renal disease? Let us turn first to the methods of testing. Formerly, heat and nitric acid covered the ground, but it is very easy to show that small amounts of albumin escape detection by this means, especially if too much of the acid is poured in. Then there were proposed from time to time numerous tests for showing traces of albumin until now we have a host of them. Of these, that of floating the urine on the nitric acid is probably the most frequently employed, and is on the whole the most useful. But, I object to these forms of tests for routine work for two reasons, neither of them on the ground of their delicacy of reaction. First, to use a test intended to show a trace is not proper, because to come at our knowledge of the urine we want to know if it contains a quantity of albumin; this knowledge boiling and acidulation give us; and at the same time it gives other information which I consider essential to the proper understanding of the specimen before us. Second, I have found all the trace tests faulty, in that occasionally, but rarely, they give a broad ring of apparent albumin, when none of that material is present. To determine the presence of albumin, in all your routine work, first boil the urine, then acidulate with a very small amount of nitric acid—and I invariably boil again; the flocculation which occurs if albumin is present—I would prefer not to speak of it as clouding, for clouding is not charac-

teristic, but flocculation is—nothing else imitates it. To judge of the quantity of albumin make it a rule to have the test-tube stand a fixed number of hours for every specimen. Thus can be arrived at by a time-standard, the relative proportion of albumin, which, if not absolute, contains each time the same variation.

To test for minute traces of albumin I have found no way so satisfying, and I believe so accurate, as the following: Fill a medium-sized test-tube less than half-full of urine, see that it is acid, using acetic acid if necessary for the purpose, then incline it sharply, and with an alcohol lamp with a very small flame boil the thin layer—the top layer—of the urine. When fully boiled bring the test-tube slowly, steadily to the upright position. If there is albumin in the top layer the supernatant urine will be flocculent, while the urine in the lower part of the tube will remain clear. If there still exists doubt there is another procedure by which albumin can be shown. I have made two records of success by the method, in both instances very important at the time. The means I allude to is by taking large quantities of urine—the more the better—acidulate it, and give it a prolonged boiling. On standing, the great bulk is sure to show positively the presence or absence of the albumin.

Recurring to the question of the value of albumin as an indication of renal disease, I have recorded very many instances of albumin without urinary-apparatus disease. Not a few of them were specimens containing a vaginal discharge, which itself was not purulent. In one of these instances the vaginal discharge contained the very largest number of spermatozoa I have ever seen in urine. The sperm fluid furnished the albumin. It was an example merely of selecting urine at an inopportune moment. I need not allude to numerous conditions in which albumin is not an evidence of renal disease.

Turn now to the heading, casts. If albumin raises, as I have said, an almost ineradicable suspicion of renal disease, casts alone or with albumin, too often doom a patient to Bright's disease. Too often the patient is told of and doomed to Bright's disease, after the careless boiling of urine in a test-tube. The nervousness resulting to the patient is frightful.

But, I wish to make the statement that casts are not Bright's disease. Casts, and something besides, are necessary for that diagnosis. I would make the statement that casts are not Bright's disease, if it did not pull the very foundations from under us, or else throw us off any sure resting place. Please, too, to remember in your judgment of me that I promised to speak mostly of exceptions, that my title to-night might be read, "The rules of urine examinations looked at from the reverse side." But,

it is yet, nevertheless, true that casts come in the urine when nephritis does not exist, and the casts may continue to come for some time.

We will speak of these exceptions after a little, but let us turn first to the finding of casts. Nothing is easier to find and define than an epithelial or granular cast, provided a few simple precautions have been properly taken. But I am convinced that casts, and with them renal disease, are not infrequently overlooked, and that, too, in a most insidious form of the disease, contracting kidney, from the want of a pretty sure method of procedure for discovering them. Let me speak of a way I have employed which serves well, allows of rapid work, and has brought success so often that I prize it highly. Everyone is ready to employ it without further preparation. How many will find it useful I cannot predict. It can be best described as a trick of illuminating the field of view. No one need start unless provided with a half-inch and a quarter objective, rigged on a double nose-piece. Turn now the mirror to give oblique light; or, even better, shade the light with the hand so that the field looks nearly or quite black. You will be surprised at seeing things invisible with the straight ray or with full light. A tube-cast previously as invisible as a clear glass rod held in the bright light will now, in the oblique ray, shine along its border as gloriously as a cloud near the setting sun. Casts which are not to be discovered by the higher power are plainly visible to the low power with oblique light. Field after field can be scanned with certainty and rapidity. Furthermore, who has not puzzled over the string of mucus, and wondered whether it was a hyaline tube-cast or not? Strings of mucus disappear under the low power and oblique light, while the rounded tube-cast is conspicuous. The mucus string is too flat, has no refracting body to be illuminated.

In conclusion I feel that I cannot be fairly charged with any crimes of omission, for I promised to speak only of things picked from my note-book, and only of a few of them. That I am open to the charge of crimes of commission, that I have found things in urine different from yourselves, and that I have interpreted others differently, I make no doubt. Please, however, accept kindly my contribution as I have found it. *April* 26, 1888.

4. *Malignant growth of kidney.*

Exhibited by Dr. I. Leopoldt.

I present to-night specimens from a case of malignant growth of left kidney, on whom the operation of nephrectomy was performed by Dr. John B. Deaver. The following is the history, as far as I was able to obtain, as the patient was very reticent regarding her life, and refused for some time to believe that she had any trouble in the shape of a tumor.

On March 11, 1880, was called urgently to see Mrs. A. S. T. Found her badly frightened by a profuse hæmaturia, and elicited the following history: Widow, æt. 41½ years, married twice, no children, several abortions, and, from what I learned later, I infer they were the result of her own exertions; family history good. Six years ago had a vesical calculus removed, and says that at that time her physician told her she was getting a tumor, which she refused to believe; she still continued at her work. Present condition pale, slightly emaciated, passed no urine for twenty-four hours, after which she then passed a large amount of bloody urine, containing large clots, the blood amounting to a little over one pint. On examining the bladder no calculus was found, uterus healthy, slight anteflexion. On examining abdomen found a large, hard mass, tender on pressure, situated anteriorly, and on left side, filling up the left lumbar, and extending into the inguinal to about one and a half inches above Poupart's ligament, and toward median line, in hypogastric and umbilical regions, two inches to the left of linea alba. The tumor was hard, was slightly movable. The whole mass could be grasped in the hand, and gave no evidence of fluctuation.

Heart sounds weak, a murmur was heard, but could not definitely time it. A diagnosis of malignant growth of left kidney was made, and patient informed of it; ordered her to quit work, rest in bed, to take ergot, and further advised a consultation, which she at the time refused.

April 2. Hemorrhage ceased, urine nearly clear, specific gravity 1022, slight trace of albumin, no casts, much epithelium from bladder, and some which appeared as if it came from the pelvis of the kidney, and blood. She improved until April 5, when another severe hemorrhage occurred, when she agreed to a consultation. Dr. Deaver was called, verified my diagnosis, and advised operation, to which she consented.

7th. Another severe hemorrhage, passed half pint of blood.

8*th*. Removed to St. Joseph's Hospital. After exertion on that day she had three small hemorrhages. I then learned that she had had one severe hemorrhage between March 12 and April 5, but had not sent for me, as she controlled it with ergot, which I had ordered should bleeding return.

13*th*. Nephrectomy was performed by Dr. John B. Deaver, assisted by Drs. Price, White, Harry Deaver, Otto, and myself, the following details of which are by Dr. Deaver.

14*th*. Death from shock twenty-four hours after operation.

Autopsy, two hours after death, performed by Drs. Deaver and Otto, and at which I was not present. Wound in good condition, peritoneal wound adherent; no hemorrhage had occurred.

Heart and right kidney removed, which I now present for your consideration:

Heart contained a chicken-fat clot, is small, left ventricle hypertrophied, cavity very small, right heart dilated.

Right kidney shows but slight changes.

Description of the operation of nephrectomy through the abdominal walls anteriorly, after the manner of Mr. Langenbuch. By Dr. J. B. Deaver.—After the patient had been etherized, also having been subjected to the ordinary antiseptic preparation, the left kidney being the seat of the growth, an incision five inches long, not extending high enough up to endanger the wounding of the attachment of the diaphragm, was made through the left linea semilunaris; the peritoneum being reached, was opened, and a large flat sponge introduced to prevent the escape of the small intestines, after which the descending colon with the medium through which it is attached to the posterior wall of the abdominal cavity, namely, the descending meso-colon, was brought into view, when its external layer was divided to an extent corresponding in length to the wound in the abdominal wall anteriorly, thus exposing the anterior surface of the growth, and giving access to the left retro-peritoneal space, which is occupied normally by the left kidney. The object of selecting the external layer is the avoidance of injuring the bloodvessels supplying the colon, thereby lessening the likelihood of necrosis of a portion of the gut. The next step in the operation was by far the most tedious as well as difficult, namely, the breaking up of the adhesions, the growth being most intimately adherent to all points of its surface, and the exposure of the pedicle, consisting of the ureter, renal artery, and renal vein. In breaking up the adhesions it required great care not to tear through the internal layer of the descending meso-colon, and the inferior layer of the transverse meso-colon,

which latter separates the left kidney from the spleen; both of which layers of peritoneum were found intact at the autopsy. When the growth was freed almost up to the point of attachment of its pedicle, a stout silk ligature was thrown loosely around the still engaged pedicle, in the event of that part of it being torn, to give immediate means of arresting any bleeding that might result (by simply tightening the ligature). After the completion of the separation of the adhesions the pedicle was grasped close to its attachment to the growth by a pair of Spencer Wells's pedicle forceps, and tied with a strong silk ligature *en masse* immediately beneath the forceps, the growth cut away and then the pedicle separated below the ligature into two portions, the vein as one and the artery and ureter the other, each of which being included in a single silk ligature. The object of separating the vessels, the artery from the vein, was to prevent the likelihood of the formation of an arterio-venous wound. The next step was the toilet, which was made with hot distilled water, the introduction of a glass drainage-tube, the reposition of the descending colon, which during the operation was drawn toward the middle line, the rearrangment of the small intestines in their proper position, as well as the great omentum, the introduction of six deep silk sutures, and the dressing of the wound. *May* 10, 1888.

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5. *Atrophied kidney, containing peculiar calculi.*

Exhibited by Dr. D. J. M. Miller.

The patient from whom these specimens were taken was an Italian, æt. 30 years, who was admitted to the Episcopal Hospital in a semi-unconscious condition on June 9. The only history obtainable was the statement of the person bringing him, that he had been found unconscious on the street. I did not see him during life, nor was I present at the autopsy, but from Dr. Van Pelt, the resident physician, the following facts were obtained: The patient on admission was semi-unconscious, but could be made to mutter a few words in Italian when aroused. His breathing was slow and labored, but not stertorous. There was no cutaneous œdema discernible, nor was there any collection of fluid in any of the cavities. The pupils were normal, and reacted to light. There was no paralysis. The lungs contained numerous moist râles, both anteriorly and posteriorly. The heart seemed healthy. There were no atheromatous changes in the arteries. The temperature was

96°. A large quantity of pale, limpid urine was drawn from the bladder; it contained no albumin. A microscopic examination was neglected. No change was noted until the next morning, when the signs of pulmonary œdema had greatly increased. The stupor deepened into coma, and he died at 3 A. M. the next day, or thirty-five hours after admission. There was a slight convulsion just before death. The temperature never rose to normal.

At the *autopsy* congestion of the meninges was found, with a small effusion at the base of the brain. The lungs presented the ordinary signs of œdema. The heart was healthy; there was no hypertrophy. The other organs were normal, with the exception of the kidneys, which were highly atrophied, the right weighing three-quarters of an ounce, the left two ounces. On section the right kidney was found in a high state of atrophy, hardly any of the parenchyma remaining. The pelvis, and especially the calices, were so enlarged as to constitute the greater part of the organ, which at one part was a mere membranous sac. Their cavities contained hundreds of irregularly spherical-shaped black bodies, from the size of a pea to that of the minutest granule. They were soft and easily crushed by the fingers, and contained no nucleus. Besides these there were two larger calculi seemingly composed of the same material; but close examination revealed that the soft black material was laid in laminæ upon a hard grayish-white nucleus. In the left kidney the atrophy was not marked, nor were the pelvis and calices as enlarged. In the cavities of the latter two calculi were found. They were of medium size, and the larger was very much like those described; but in the smaller the dark material was laid in laminæ upon a hard faceted brownish calculus. The nature of these bodies is not clear. They resemble very closely altered blood, yet microscopic examination failed to reveal blood-cells, while Teichman's test for blood crystals was negative; but the failure may be due to the inexperience and want of skill of the exibitor. It is unfortunate that no history of this case could be obtained, for thereby much light might be thrown upon the specimens.

Report of the Committee on Morbid Growths: "Your committee reports on the specimens presented by Dr. Miller as follows: The larger masses are formed of a hard but friable mass of grayish color, covered by a soft, darker, almost black layer. This inner mass shows under the microscope amorphous crystalline masses, and red rhombic crystals and sheaves. Chemically examined, burns with a white residue, does not respond to murexid test, is insoluble in water, soluble in mineral acids without effervescence, insoluble in acetic acid; when heated and treated

with the same it dissolves with effervescence. This proves it to be calc-oxalate. The outer covering is soft and fibrous in structure. Under the microscope it shows fibrous bands and red rhombic crystals, needles, and sheaves. It swells slightly, but is otherwise unaffected by acids, but dissolves in alkalies (KHO). The rhombic crystals and sheaves are also soluble in cold alkaline solutions, and are therefore hæmaglobin. The soft fibrous pellets and masses are evidently fibrin, and the process one of calculi, with hemorrhage into the renal pelvis."

June 14, 1888.

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6. *Tuberculosis of the kidney and ureter.*

Exhibited by Dr. J. Tyson.

The patient whence this kidney was taken came under observation September 18, 1885. A little more than a year previous he began to have pain in the neck of the bladder at the end of micturition, which also became more frequent, although he was not certain that the two symptoms appeared simultaneously. The pain soon passed away, but at times it would last longer. In October, 1884, he first noted a little blood in his urine, also at the very end of the act of micturition, and amounting to less than fʒss. His family physician examined his urine, and said he had catarrh of the bladder, and that there was no renal disease. The hemorrhages, which occurred first about once in three days, gradually became less frequent, but on September 18, 1885, they had again become as frequent. He was rising about three times a night to pass water, always with pain in finishing. In addition paroxysms of pain, independent of micturition, now occurred. The urine at this time contained about one-eighth its bulk of muco-pus, about the same amount albumin, but no casts, no blood. Walking and driving aggravate his symptoms. December 15, 1885, Professor Agnew sounded him for stone, with negative results.

Under treatment his subjective symptoms improved, and the hemorrhages, never large, became less frequent. Pus continued in his urine in varying quantities, generally ranging from one-fifteenth to one-tenth of its bulk, but on the whole it may be said that the bladder symptoms gradually abated. He had occasional attacks of feverishness, which he himself regarded as malarial, and he continued to attend to business with more or less regularity until about the middle of December, 1887. During this time he was assured that the kidney was not diseased, that the symptoms

were those of a bladder affection, in which the disease was believed to lay. At the time mentioned he took cold, and became confined to his house. He recovered partially to again relapse. His cough never entirely disappeared. Diarrhœa set in, and was only partially controlled. These symptoms persisted with very gradual increment. In May it was evident that softening of the left apex was beginning to take place, and that the diarrhœa was tubercular. In July he went to the country, where he died early in September, under the care of Dr. Sinkler. During the last two months of life the liver rapidly enlarged. Three or four days before death there was a hemorrhage from the urethra, accompanied by bubbles of gas.

The *autopsy* was made September 5. Emaciation was extreme.

Thorax: There were decided adhesions between the left lung and the corresponding thoracic wall, but on the right side they were but slight at the apex and between the base and adjacent diaphragm. There was a cavity in the upper lobe of the *left lung* fully four inches in diameter, while its remainder was pervaded by numerous tubercular broncho-pneumonic foci. There was a scar-like depression on the apex of the *right lung*, and tubercular broncho-pneumonic foci throughout it. The *heart* was normal. The *liver* was a typical amyloid one, fully twice the normal size. The *spleen* was amyloid. The *right kidney* was at least twice as large as the normal organ, and on section exhibited the appearance shown by the specimen, that of a typical caseous tubercular kidney. There were seven cystic foci, corresponding to as many pyramids, filled with soft caseous tubercle. The corresponding ureter was also infiltrated with tubercle. This kidney was adherent to the adjacent viscera. The *left kidney* was also one-third larger, and amyloid, but not tubercular. There were adhesions between the bladder and other pelvic viscera, but the *bladder itself*, except that the mucous membrane was slightly hyperæmic, was normal. The prostate was not enlarged. The *ileum* contained numerous disseminated miliary tubercles and tubercular ulcers, and the large intestine was shrunken, and adherent to the bladder. The mesenteric glands were tubercular. The most interesting and important feature of this case was the total absence of symptoms pointing to the kidney itself, and the presence of all the signs usually relied upon as indicating a bladder affection. It was not until toward the termination of the case that the certain signs of tubercular disease elsewhere raised the suspicion that there was tuberculosis of the bladder and kidney, confirmed as to the latter and ureter, but erroneous as to the former. *October* 25, 1888.

7. *Tuberculous kidneys from a child.*

Exhibited by Dr. G. E. de Schweinitz.

These organs were removed, *post-mortem*, by Dr. Eckman, from a patient in the ward of Dr. Ashhurst, in the Children's Hospital. E. S., a boy, æt. 9 years, had been an inmate of the hospital for one year, and the subject of coxalgia for two years. The disease had developed after an injury. Excision of the hip had been performed, but there was, later, extension of the process (tubercular) into the acetabulum, with pus accumulation in the cavity of the pelvis, and persistent suppuration. No cough or sign of pulmonary trouble had developed, but stubborn diarrhœa, with progressive and huge enlargement of the liver, resulted. The quantity of urine was unduly great, but the sediment was negative. At the autopsy (confined to the abdominal cavity) the liver and spleen were found to be amyloid, and both kidneys showed a beautiful, circumscribed tuberculous process, most pronounced upon the right side.

November 8, 1888.

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8. *Suppurative nephritis following abortion.*

Exhibited by Dr. J. C. Wilson.

Mary R., æt. 40 years, married; born in Ireland; a domestic. Admitted to Philadelphia Hospital, July 3, 1888. Family history good. Patient has always been a healthy woman; has had nine children, and one miscarriage, which occurred on the 15th of March, 1888; no history of addiction to alcohol or of venereal trouble.

On March 15, 1888, the patient aborted in the fourth month of pregnancy, at which time she lost a large amount of blood; she remained in bed for two weeks. On the day she got up she had a severe chill, followed by a fever which lasted about two days, and was accompanied by severe pain in the back and left lumbar region. This pain was shooting in character, and extended from the back forward and downward to the region of the left groin. About this time she began to suffer from frequent desire to pass water and burning pain in the region of the bladder, and observed that the urine was whitish in color and deposited a thick sediment. No history of having passed blood by the bladder. The bowels had been disposed to be loose. On admission complained of constant

severe pain in the left side, extending from behind forward and downward, which was aggravated on movement, and on turning either upon the right or left side. Patient states that she is never free from this pain, and that in consequence of her suffering she has, during the last four months, lost more than forty pounds; expression of face is indicative of suffering; skin sallow, not jaundiced; tongue large, pale, and coated; no appetite; bowels inclined to be loose; pulse 102; respiration 30; temperature 101.4° F.; temperature ranged between normal in the morning and 102° in the evening; urine contained pus in varying amounts, and was always acid in reaction; upon microscopical examination nothing was found except pus cells and granular detritus; vaginal examination revealed an atrophied cervix and dilated os; there was no uterine or vaginal discharge; no pelvic tumors.

Physical examination: Lungs resonant, no râles; heart normal, first sound faint, second aortic sound accentuated; liver dulness extended to margin of ribs.

The abdominal walls contain a fair amount of fat, are somewhat relaxed, but not pendulous; percussion dulness in the left hypochondrium extending two inches and a half toward the median line, from the anterior superior spine of the ileum. This dulness extends upward, and becomes continuous with the dulness of the left lobe of the liver, and toward the left with the splenic dulness. Upon right lateral decubitus the line of dulness is shifted slightly toward the median line. Upon palpation, which elicits considerable pain, there is to be detected a tense tumor, the surface of which seems to be slightly lobulated. It is noted that the surface of the left lumbar region is somewhat flattened, both to the eye and upon palpation, as compared with the corresponding region on the opposite side.

About two weeks after admission Dr. J. B. Deaver saw the patient in consultation. Catheterization of the left ureter was successfully performed, the instrument passing easily several inches into the ureter, and discharging a thick purulent fluid, drop by drop. Upon repeated attempts neither Dr. Deaver nor myself succeeded in passing the instrument into the right ureter, although it was not difficult to engage its tip in the outlet.

Patient was operated upon by Dr. Deaver on the 24th of July, the kidney being reached by an extensive incision in the left lumbar region. About four ounces of thick, creamy pus were evacuated from two abscess cavities. The inflammatory thickening of the capsule and of the surrounding tissues was so great that the idea of nephrectomy was abandoned. The drainage-tube was introduced, and the wound dressed;

intractable vomiting followed the operation; the temperature steadily rose, reaching on the following morning 104.4° F. Death occurred about twenty-four hours after the operation. During this interval small amounts of purulent urine were withdrawn by means of the catheter; pus was discharged through the drainage-tube in the wound.

No post-mortem examination was permitted by the friends of the deceased, but the kidney was subsequently obtained outside of the hospital under circumstances which precluded any investigation of the other organs. The left kidney was decidedly enlarged and thickened, measuring before being placed in alcohol about seven inches in length by four in breadth, and two and a half in thickness. Its capsule was enormously thickened, and matted to the adjacent structures. The ureter, which had been cut through close to the surface of the kidney, was at that point about an inch in diameter. Upon separation of the capsule, which was readily detached, the kidney was found to be markedly lobulated, and at one point on its outer surface there was a small abscess cavity filled with white, creamy pus. The whole structure of the organ was extensively altered, the line of demarcation between the cortex and medulla being obliterated. A series of irregular pus cavities, varying in size from a pigeon's egg to a small marble, occupied the kidney substance. These cavities in some instances communicated with each other. The pelvis of the kidney and the papillæ were the seat of extensive new growths of fibrous tissue.

No tubercle bacilli were found either in the pus or the scrapings of the walls of these cavities. *November* 22, 1888.

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9. *Anomalous ureter.*

Exhibited by Dr. G. H. Chambers.

The specimen is an anomaly of a ureter. It was removed from a body in a dissecting-room. The subject was that of a male, colored, middle age. The anomalous ureter is attached to the left kidney.

The left kidney is furnished with two ureters, one of which is somewhat larger than the other in thickness. The larger of the two originates from the middle of the hilus of the kidney, but the smaller ureter arises from a point to the inner side of the kidney, and below the origin of the larger one. The distance between the points of origin of both ureters is about one inch. The two ureters pass from the kidney for a

distance of about seven inches, they then unite to form a single ureter of the length of about five inches, which enters the bladder in the usual way.

Whether the smaller ureter can be regarded as merely a supernumerary one, or whether the two together may be considered to form strictly a double ureter, is, to my mind, undecided. The right ureter is normal.

There appears to be only a single pelvis for the anomalous ureter.

There were no anomalies in the other organs.

December 13, 1888.

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10. *Acute nephritis in typhoid fever.*

Exhibited by Dr. W. Osler.

Specimen from a case of typhoid fever, with renal complications, were exhibited. The patient, a woman, æt. 25 years, was admitted about the eigthth day of the disease. She had delirium, very rapid pulse, but very slight intestinal symptoms. The spots were well marked. Temperature was never above 104°, and on two occasions it sank to subnormal. The urine was scanty, and contained a large quantity of albumin, many granular tube-casts, and a few blood corpuscles; no large amount of blood. Death took place on the thirteenth day. Ulceration of the intestines was extensive, and the edges of the ulcers were hemorrhagic. The kidneys were large, and together weighed twelve ounces. The cortices were pale, with only the large veins dilated; marked contrast between the cortical and pyramidal portions. Microscopical examination showed the convoluted tubules much swollen and filled with granular detritus. There were many tube-casts in the tubules of the cortex and in those of the pyramids.

February 28, 1889.

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11. *Very small cystic kidney; death from uræmia* (?).

Exhibited by Dr. H. M. Fisher.

Celia C., æt. 25 years, single, housemaid, was admitted to the Episcopal Hospital November 29th, in an unconscious condition.

The resident physician, Dr. B. B. Reath, handed to me the following

brief record, which was kindly furnished to him by Dr. Noble, who had been called to see the patient before her removal to the hospital:

"Patient had washed clothes on the 26th, and had been exposed to the cold and wet. She was passing through her menstrual period at that time. On the night of the 27th her menstrual flow suddenly stopped, and she had sick stomach and headache. She was given hot teas and Seidlitz powder, and hot bottles were put in her bed that night." On the morning of the 28th Dr. Noble was sent for again, on account of the "girl being in fits." The convulsions appeared to him to be of an hysterical nature, as consciousness was not abolished in them, and she answered questions that were put to her during the convulsions.

Morph. sulph. gr. ¼ was given by hypodermic injection. Chloral grs. x., sodii bromid. grs. xx., and elixir. ammonii valerianat. ʒii., ordered.

In the afternoon of the same day the doctor saw the patient again. She was then unconscious, and had had seven or eight epileptiform convulsions. Urine was found to be albuminous.

Upon her admission to the hospital her pupils were contracted, skin hot and dry. Temperature 103°; pulse 154, strong and full. Patellar reflexes absent, but reflex contractility still maintained. Slight œdema of legs. No heart-murmurs could be detected. Impairment of resonance over lower third of both sides of chest, and bronchial breathing at lower angles of scapulæ, with subcrepitant râles in these regions. Urine pale yellow, sp. gr. 1022, neutral in reaction, and loaded with albumin.

Enema administered; wet cups were applied to loins; hot vapor bath given, followed by hot digitalis fomentations. Skin did not act freely in bath, and patient remained unconscious on the morning of the 30th, when I first saw her. She could no longer swallow; her pulse was about 200, and her breathing was hurried and irregular.

She was catheterized three times, and only thirteen ounces of urine in all were drawn off. Ordered atropiæ sulph. gr. $\frac{1}{100}$ hypodermically, every four hours, hot vapor baths every two hours.

Patient, however, continued to grow weaker, and died December 1st, at 3 A.M.

Post-mortem examination, thirteen hours after death.—Posterior portion of lower and middle lobes of right and of lower lobe of left lung non-crepitant, and *tissue does not float*, while anterior portions are somewhat crepitant. Cheesy nodules are found in very considerable numbers in lower lobes of both lungs, surrounded by infiltrated lung tissue. The appearance of these cheesy masses seems to point, however,

to their not having been of very recent formation, and the tissue not occupied by them is plainly hepatized. In some places there are patches of gray hepatization.

Old pleuritic adhesions. At apices scars from old cheesy deposits seen.

Heart: Left ventricle hypertrophied. Right ventricle dilated and empty. Both auriculo-ventricular valves contain chicken-fat clots.

Kidneys: Right weighs one and a quarter ounces. Capsule very adherent, and tissue granular. Almost complete cystic degeneration, cortical portion being about one-quarter inch in diameter, and medullary portion almost absent. Left kidney weighs five and a half ounces, and divided into two lobes. Tissue granular. Capsule adherent. Several cysts in cortical portion.

Right ovary cirrhotic; left contained a cyst about the size of a pigeon's egg.

Bladder empty. *February* 28, 1889.

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12. *Horse-shoe kidney.*

Exhibited by Dr. J. H. Musser.

This specimen was removed from the body of a man who died of carcinoma. After opening the abdomen the transverse portion could be seen as a tumor over the vertebral column. Had the patient been carefully palpated over the abdomen during life its presence could have been recognized, and thus possibly be taken for some diseased structures.

It is a perfect specimen. The connecting band of the arms of the shoe is normal renal structure. The bloodvessels and ureters are distributed in a natural way to the pelves, which are normally situated. The kidney was situated with the arms pointing downward.

March 28, 1889.

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13. *Vesical calculus.*

Exhibited by Dr. A. A. Eshner.

Patient was admitted to hospital December 22, 1888, with a history of difficulty and increased frequency of micturition, associated with pain. The day before he had suddenly been seized with vertigo, and had

fallen. For six months his feet had been swollen. Any venereal history was denied, though patient had on several occasions been treated for some obstructive disorder of micturition. Examination of the urine disclosed the presence of pus, mucus, phosphates, and blood; the urine, however, sometimes being clearer and freer from abnormal ingredients than at others. Patient was again admitted on March 29, 1889, having had "a rush of blood to the head." His condition was one of great debility. The temperature was 104.° There was incontinence of urine, analysis of which revealed the presence of pus-corpuscles, crystals of triple phosphates, and a doubtful trace of albumin. Under treatment by rest and stimulation the temperature gradually declined, but on April 6th the patient succumbed to exhaustion.

At the *autopsy* a large ovoidal calculus, probably of phosphatic composition, of the size of a pullet's egg, was found lying in the bladder, the walls of which were greatly thickened, and the lining membrane roughened and ulcerated.

The *ureters* were widely dilated, and their walls thickened. The same conditions existed in the pelves of the kidneys, the lining membrane of the right presenting evidences of having been involved in a membranous inflammatory process. The kidneys were of about the normal size. The capsules were somewhat adherent and thickened. The surfaces of the organs were irregular and lobulated. On section the parenchyma showed fatty changes in parts. *April* 11, 1889.

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b. FEMALE ORGANS OF GENERATION.

14. *Bilateral dermoid cysts of the ovaries.*

Exhibited by Dr. G. Dock.

The specimens were removed from a multipara, æt. 30 years, whose history is unimportant from our present point of view. They are the two ovaries with the tumors about to be described. The right ovary is about the size of a duck's egg. The surface for the greater part is smooth, part showing the usual cicatrices of the normal ovary. Most of the bulk is taken up by a cyst the size of a hen's egg, the walls of which are thin, the surfaces smooth, and the cavity of which is filled with a substance resembling butter, both macroscopically and microscopically, and

numerous tightly rolled-up fine blonde hairs. In the wall of the cyst adjacent to the ovarian tissue is a small cyst, the size of a pea. The ovary contains three false corpora lutea. The left ovary is larger and more irregular in outline. Most of it is taken up by a thin-walled cyst, the size of an orange, which is not simple, but formed by the opening into it of two or more small cysts. It contained clear watery fluid. Near it are several cysts of small size, filled with a tough brownish mass. Next is a cyst the size of a walnut, with thin and smooth walls. The cavity is filled with light-brown hair, and grayish granular and greasy material. At one end is a tooth, resembling in size and shape a deciduous canine tooth. Enamel seems to be entirely absent, and on one side is a crater-shaped defect. The tooth is growing into the thin wall of the cyst, which at that point presents no peculiarities of structure. Adjoining this is another cyst, also containing hair and fat. The walls of these cysts are not so characteristic as usual, the various layers of the true skin not being present in any very great extent. The specimens were rather spoiled for histological examination, but we can still see the large sebaceous glands, and apparently remnants of sweat glands. The buttery substance is shown to be pure oil, and the granular mass oil mixed with broken-down epithelial cells. Cholesterin entirely absent. The other cysts mentioned are in all probability follicular cysts.

October 13, 1887.

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15. *Uterus and appendages from a case of peritonitis, etc.*

Exhibited by Dr. G. Dock.

The following is all that is known about the antecedent history of the person from whom these specimens were taken: Mrs. N., colored, æt. 25 years; married eight years. One living child, thirteen years old. Menstruation said to have been regular. Two weeks before admission to University Hospital patient felt severe pain in abdomen, lasting two or three hours, and followed by slight pains at times ever since. Abdomen was swollen and painful. A diagnosis of pelvic peritonitis was made. Patient died five days after admission.

On opening the body the anterior belly walls were found in close contact with, though not adherent to, subjacent structures, the surfaces dry. Parietal peritoneum thickened and injected. The well-preserved omentum covered the lower abdominal cavity, being bound down by firm

adhesions to the wall of the iliac fossa, and to the coils of small intestine in the pelvis. On separating the adhesions the under surface of omentum found covered by a thick layer of lymph, the fat tissue much injected and infiltrated. The intestines were bound together by masses of lymph, and floated in a large collection of thick, extremely fetid, yellow pus.

The *thoracic* organs presented no noteworthy lesions.

Spleen of normal size. Adherent on all surfaces; capsule thickened; tissue pale, soft, and friable.

Liver weighed two pounds. Left lobe firmly adherent to diaphragm; capsule thickened and corrugated, cut surface bloodless, pale yellow; acini not well marked; gall bladder and ducts normal.

On separating the coils of adherent intestines the full bladder and uterus were seen almost filling in the entire true pelvis. The broad ligaments and tubes were not in their normal position, and the whole pelvic contents were removed *en masse* for dissection.

Bladder contained a half pint of turbid urine; serosa covered with fibrin and pus; no lesions of mucosa.

Vagina roomy; rugæ obliterated; mucous membrane smooth and pale; vulva showed no lesions.

Uterus measured 12 cm. in length, 8 cm. in width at level of tubal insertion. Cavity 10 cm. long, 6 cm. wide. Wall 2 cm. thick at fundus, 2.5 cm. anterior wall of body, and 1.5 cm. at cervix. Muscular wall soft and pale; in the gaping vascular channels, cut across, were plugs of pale-reddish fibrin. Mucous membrane thickened, especially in cervical canal, and intensely congested in body. In the right anterior wall was a mucous polyp, the size of a cherry, sunk in the thickened mucous membrane.

The whole surface of the uterus was covered with thick and firm lymph. On dissection the right ovary was found lying close to posterior surface of uterus, at base of body. The right tube, twisted and dilated, was folded back so as to cover in the ovary; the terminal, most dilated part, on which no traces of fimbriæ could be seen, reached the level of the lowest part of the cervix. The left ovary was covered and fastened down in a manner resembling the right, but the tube was not so much dilated. In the extreme end of the latter was a cyst the size of a cherry, containing clear watery fluid. This ovary contained a clot the size of a walnut, in a smooth-walled cavity. There were no recent corpora lutea in either ovary. In the right broad ligament, near the uterus, was a diffuse swelling, during examination of which pus escaped from the artery leading toward it. This swelling proved to be caused by a series of small

cavities containing very fetid purulent fluid. One of the cavities had burst into the artery, probably during examination. In the wall of the uterus, just below the right tube, and not communicating with it or the other cavities described, was a small pocket containing fetid fluid and necrotic muscular tissue.

Much of the value of the case is lost for want of details regarding the clinical history. Still, the lesions are so well marked as to be in themselves very instructive. The course of the processes was probably about as follows: Chronic endometritis and metritis, inflammation propagated to tubes and pelvic connective tissue. From these, lighting up of a general septic peritonitis.

Concerning etiology but little can be said. No doubt many of these cases are of gonorrhœal origin, and the character of the endometritis in this case would be considered by some as indicating that cause. The fetid liquid in the abscesses was examined for gonococci, but though full of bacteria, mostly long rods, single or in chains, no diplococci could be found at all. This, of course, does not exclude gonorrhœa from among the causative agents. There is no evidence that there had been a recent impregnation. *May* 10, 1888.

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c. MALE ORGANS OF GENERATION.

16. *Alveolar sarcoma of testicle.*

Exhibited by Dr. T. C. Wescott.

The patient, a man æt. 54 years, father of thirteen children, with no venereal, and apparently a good family history. About a year ago he had received a kick in the privates from a man with whom he was scuffling, but had attached no importance to the injury, and soon forgot it. About five months ago he noticed that the left testicle was increasing in size. The right gland had begun to enlarge about two weeks before, according to his own statement. Pain was not felt except as an occasional twinge. The scrotum presented very much the size and appearance of a bull's scrotum, and the vaginal tunics both contained liquid. A section cut from the growth in the cord gave the microscopic character of an alveolar sarcoma, which I feel inclined to believe it, despite the advanced age of the patient and its apparent rapidity of growth. *December* 8, 1887.

17. *Tuberculosis of the testis.*

Exhibited by Dr. G. Dock.

This specimen was removed by Dr. Keen from a patient in the practice of Dr. E. R. Stone, to whom I am indebted for the opportunity of presenting it.

The clinical notes of the case, kindly furnished by Dr. Stone, are as follows:

W. M., æt. 33 years; father died of some disease of liver in early manhood; mother had chronic diarrhœa for several years, and died, at age of thirty-six, of diarrhœa and wasting. Dr. Pepper verified probable diagnosis of tubercular ulceration. No other case of tubercular disease among the near relatives.

Patient is an only child. Previous health good until age of 22, when he had remittent fever, with double plastic pleurisy. After recovering from that had several attacks of malaria. In one of these, about the spring of 1883, one epididymus became inflamed, and afterward the testicle itself was involved. An abscess formed, which was opened. The testis remained hard and swollen, and a sinus, discharing a small amount of thin pus, persisted until the organ was removed.

The entire absence of any venereal disease is perfectly established. Patient has no lung symptoms. General health much improved since the operation.

Dr. Keen makes the following addition to the note: Testicle removed June 21, 1887, by W. W. Keen, with antiseptic precautions. Combined tubular and capillary drainage. Entirely well in seven days, and has been perfectly well ever since.

On examination the organ is found enlarged, especially the epididymis. Surface smooth. In the middle of the epididymis there is a sinus with infiltration around its orifice.

The head and tail of epididymis each contain large cheesy masses, and their walls are thickened. Extending into the testis proper from the mediastinum is a cheesy mass the size of a filbert, with irregular, thin fibrous walls. Near it is a small focus the size of a pea, and in the vicinity numerous foci of pin-head size. The infiltration extended but a short distance beyond the tail of the epididymis, in the vas deferens. The cellular and vascular part of the cord not affected. The rest of the parenchyma, on section, shows numerous roundish bodies, from one to two

mm. in diameter, scattered through it. They are firm, have a pearly appearance on section, and some show minute cavities in the centre. Examination of the caseous material shows a few tubercle bacilli.

February 9, 1888.

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18. *Hæmatocele.*

Exhibited by Dr. F. A. Packard.

This specimen was removed by Dr. Ashhurst, at the Pennsylvania Hospital, March 7, 1888, from a patient æt. 18 years, born in England. He is intensely stupid, and can give no history as to the first time of appearance of tumor. On admission the right testicle was much swollen, tender, with a somewhat doughy feel, and a sense of semi-fluctuation. No light was transmitted. He gave a history of long-enduring tumor (painless) in the right testicle. Three weeks ago he noticed the tumor rapidly swelling, after he had been running hard. Shortly after he began to have severe sharp pain in the testicle and lower portion of abdomen. Thinks that he has not lost flesh or strength. Prior to its removal a trocar and canula were thrust into the tumor, with the escape of a few thick drops of grayish and streaked fluid. The tumor is seen to be covered by the tunica albuginea. On section there is found to be a hæmatoma with rough walls. The contents of the cyst being apparently a softened broken-down blood clot. *February* 9, 1888.

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19. *Tumors of the testicle: two cases.*

Exhibited by Dr. H. R. Wharton.

Case I. *Sarcoma of Testicle.*—Pat. B., from whom the tumor of the testicle was removed four years ago, noticed a swelling of the right side of the scrotum. This was tapped twice, and caused him little pain. In October, 1888, the tumor was again tapped, since which time it has been painful, and has increased much in size. The patient was admitted to the University Hospital, and the tumor was removed by Professor Ashhurst, on February 10, 1889. Section of the tumor after its removal showed that it was made up of several large cysts containing a

dark-colored fluid, and at points the tumor contained masses of cartilaginous material. The growth is probably a spindle-celled sarcoma, and, according to Butlin, cysts are most commonly met with in this variety of sarcoma. The presence of cartilaginous masses at different parts throughout the growth is also in favor of its being a spindle-celled sarcoma, as chondrification is also common in this variety of sarcoma.

Case II. *Tuberculous Testicle* removed from a patient in the University Hospital, æt. 29 years, by Professor Ashhurst. The patient, during the summer of 1888, noticed right testicle enlarging. The enlargement was gradual, and a few months ago an abscess formed, perforating the skin of the scrotum. On section the body of the organ is seen to be much enlarged, and at points the tissues are broken down and forming abscesses. Tuberculous disease of the testicle is most common in young adults, and generally begins by the formation of an indolent swelling of the globus major of the epididymis, and is frequently associated with tuberculous manifestations in other portions of the body. The tuberculous masses are apt to break down, forming abscesses which discharge at various points through the skin of the scrotum. This was the case in this specimen. *February* 28, 1889.

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20. *Tuberculous testicle.*

Exhibited by Dr. H. R. Wharton.

The testicle was removed from a boy, æt. 4 years, a patient in the Children's Hospital, who was admitted in August, 1888, with arthritis of left knee-joint, which had gone on to suppuration.

The left knee-joint I excised in September, and the patient did well after the operation. About this time it was noticed that the right testicle was enlarged and hardened, and this enlargement increased gradually, and all the symptoms pointed to a solid tumor of the testicle. Coincident with the growth of the testicle the patient presented a localized inflammatory swelling, involving the periosteum and tibia of the right leg.

The tumor of the testicle was supposed to be tuberculous, and I removed it on January 2, 1889.

On section of the testicle it will be seen that the body of the organ is enlarged, and that at certain parts it has undergone caseous degeneration.

This specimen is of interest when we consider the age of the patient, for in a large experience with children suffering from scrofulous or tuberculous affections this is the first time in which I have seen the testicle involved, although I have seen a number of cases of the affection in adults in connection with pulmonary phthisis or tubercular affections of bone.

True tubercle of the testicle, according to modern pathologists, originates in the interstitial areolar tissue. *March* 28, 1889.

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d. MAMMARY GLANDS.

21. *Recurrent sarcoma of the breast.*

Exhibited by Dr. H. R. WHARTON.

This was removed from a patient in the University Hospital, by Professor Ashhurst. The original growth, which was a very large sarcoma, weighing about seven pounds, was removed six months previously, and was presented before the society at that time.

A careful examination of the axilla, on the occasion of both operations, failed to reveal any involvement of the axillary glands.

December 22, 1887.

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22. *Soft cancer of breast.*

Exhibited by Dr. E. MARTIN.

Woman, æt. 38 years, unmarried. One year ago a lump appeared beneath the skin on the upper aspect of the breast. Lancinating pains, and much anxiety on the part of the patient. Removal, and microscopic diagnosis of adenoma. Three months ago reappearance of tumor in seat of former operation. Great pain. Breast apparently healthy. Nipple not retracted. Microscopical examination shows typically soft cancer.

January 26, 1888.

23. *Scirrhus of the breast.*

Exhibited by Dr. H. R. Wharton.

This was removed from a woman, æt. 50 years, by Professor Ashhurst, at the University Hospital, February 12, 1888. The patient first noticed the tumor in the left breast in December, 1887, and the principal point of interest in this case is the rapidity of growth in the tumor in a woman at this age, for in patients over 50 years of age many of these tumors grow slowly, and are often accompanied by little pain, and tend to present the characteristics of atrophic scirrhus.

February 23, 1888.

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24. *Tumors of the breast; two specimens.*

Exhibited Dr. H. R. Wharton.

Case I.—A large tumor of the breast removed by Professor Ashhurst from a woman, æt. 43 years, a patient in the University Hospital. This patient had an ovarian tumor removed by Professor Goodell, eight years previously, and first noticed the present growth in the right breast June 17, 1887, when it was about the size of an egg. Since that time the tumor has steadily increased in size, without any tendency to ulceration, until it has attained its present dimensions.

The tumor after removal weighed seven and a half pounds, and it is probably a cystic sarcoma.

Case II.—Tumor of the breast removed by Professor Ashhurst from a woman, æt. 33 years, a patient in the University Hospital. This patient first noticed a small movable lump in the left breast in June, 1887, which gradually enlarged, but was accompanied with little pain. During March of this year ulceration of the skin over the tumor occurred, and at the time of operation the axillary glands were much enlarged.

This tumor is probably a carcinoma of the breast.

June 14, 1888.

25. *Ulcerated carcinoma of the breast.*

Exhibited by Dr. H. R. Wharton.

Margaret S., æt. 50 years, first noticed a tumor in right breast three years ago. This increased slowly in size, and its growth was accompanied with pain. In July, 1887, the growth became ulcerated, and from this time on until the time of operation numerous hemorrhages occurred from the surface of the ulcer, some of them being quite profuse. In October she presented herself at the University Hospital, having just suffered from a very profuse hemorrhage, the effects of which she showed in a very marked degree. Although the case was not a favorable one for operation, as there was marked involvement of the axillary glands and infiltration of the skin around the tumor, in view of the frequent and profuse hemorrhages it was considered advisable to remove the growth. Professor Ashhurst, under whose care the patient was, removed the breast and a number of enlarged axillary glands, and since the operation the patient has done fairly well, although the wound is not yet *healed*.

I also present specimens of carcinoma of the breast as card specimens, as I am unable to get a history of them. *September* 27, 1888.

VI. THE NERVOUS SYSTEM.

1. *Case of neuritis, superinduced by caries of eighth and ninth dorsal vertebræ.*

Exhibited by Dr. J. M. Vincent.

June 28, 1886, I was called to see F. K., railroad engineer, æt. 41 years. He complained of pain in right thigh, and in right side. The point of greatest intensity was just over the crest of the ilium and under the twelfth rib, whence it was reflected upward and posteriorly toward the spinal column, and downward, at times following the course of the sciatic, and at other times that of the anterior crural nerve.

At these points the pain was increased by pressure.

Careful inquiry into his history did not reveal anything that was calculated to throw much light on the case, except to show how obscure and masked it was from the very start.

He had been examined and treated by many physicians of ordinary skill and ability, for pleurisy, for gall-stones, for congestion of the liver, etc.

After a careful examination both of his condition and history, and finding nothing which in my opinion would at that time justify anything else, I made a diagnosis of intercostal and sciatic neuralgia, and treated him accordingly. But not being able to persuade him to take a much needed rest, the treatment did not avail much, and he, growing weary of it, passed into other hands.

October 28. After undergoing treatment by several other physicians for rheumatism, lumbago, etc., he returned in a very much worse condition than when I last saw him. I then put him on ol. morrhuæ and electricity, but through persuasion of some of his friends that more might be done for him elsewhere, he again passed out of my hands.

I then saw nothing more of him till the March following, a day or two before a journey to Philadelphia to get an opinion at Jefferson Hospital. At this time he was convalescing from a very severe abscess on the right thigh, following use of hypodermics, and was very much emaciated and reduced.

In Philadelphia he was seen by several physicians, and the case was diagnosticated as one of neuritis, an opinion expressed in a lecture to the class by Professor Bartholow.

The following notes were made on the case when examined by Dr. S. Solis-Cohen, then chief of the medical clinic in Jefferson Medical College Hospital, on the 10th of March, 1887:

Present condition.—Constant pain in right hypochondrium and epigastrium, also in dorsal and lumbar regions of spine. General tenderness over spinal column, especially dorsal. Great tenderness over the course of the sciatic nerve. Shooting pains over region of chest, extremely sensitive to pressure on right flank over last rib. Great hyperæsthesia. Wasting of muscles of right leg and thigh, attended with loss of power. No ataxia. Right side of chest much wasted. Has lost fifty pounds of flesh. Electric examination, right lower extremity, shows quantitative motor decline. Crural and sciatic nerves very sensitive to pressure and to current.

The treatment prescribed by Professor Bartholow was the descending current, stabile, as strong as the patient can stand. Faradism for exercise of muscles. Deep injections of cocaine. Internally, lacto-phosphate of calcium, liq. potassii arsenitis, and ol. morrhuæ.

When the patient returned home he sent for me, and wished me to again take charge of the case, and carry out the treatment prescribed.

Under this treatment he improved quite satisfactorily for about one month, as evidenced by increase in weight, recession of pain, etc.

At the end of this time he began to grow rapidly worse. The pains became more severe and more constant, yielding to nothing except hypodermatic injections of morphia.

He then became exceedingly anxious to return to Philadelphia, to see if something could not yet be done that would prolong his life without suffering so much pain.

Finally, yielding to his earnest request, I accompanied him to this city about the middle of May, when he was again seen and examined by a number of physicians in the hospital, where he was put into a private room, under the care and supervision of the physicians then in charge.

He remained here under treatment for ten days. Making apparently no improvement during that time he became very much discouraged, and anxious to go home to his family; but before doing so wished to be examined by Professor H. C. Wood, who agreed with the former diagnosis and prognosis, except that he thought the inflammation might possibly be only peripheral, in which case the prognosis would, of course, be favorable. In the main he agreed with the treatment, and recommended a continuation of the same.

But, notwithstanding all that had been done and was then being done for him, he continued day by day to grow worse, the pain becoming more constant and more severe.

About this time the pains began shifting about, becoming more intense on the left than on the right; girdle pains starting in the back, and running round both ways. Of these pains there was no remission, except under morphia, till death destroyed his sensibilities.

About a week before death paresis of the lower extremities took place, involving all of the pelvic organs, and just how far it extended upward posteriorly I was unable to determine, but I know it did not involve the stomach and respiratory muscles, for notwithstanding the great quantities of strong medicines he had taken his stomach caused him no inconvenience up to the last. Death occurred by asthenia, August 4, 1887.

Autopsy.—I first took up a section of the sciatic, then made an incision from the first dorsal vertebra to the sacrum, and on running my finger along the vertebra I discovered a very distinct roughness of the eighth and ninth dorsal, which I exsected, and found to be necrosed, as were also the ribs attached at articulations; also took up the intercostal nerves given off here; also took a section of lower lobe of right lung, which was adherent to ribs, and hepatized.

None of the other organs, so far as examined, were found to be diseased.

It is believed by some who had some knowledge of this case, that its origin was specific, though no satisfactory history could be obtained upon which to base such belief, it being stoutly denied throughout by the patient. *January* 26, 1888.

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2. *Tubercular meningitis.*

Exhibited by Dr. Berens.

Child, æt. 2 years, admitted March 3, 1888, to Children's Asylum, Philadelphia Hospital, in an unconscious condition. Temperature 100⅓°. Pulse rapid and feeble. Respirations quickened. It lay on its back, with legs flexed on thighs, and thighs on abdomen. Slight internal strabismus, which was congenital. Pupils equal. Belly scaphoid. Third day, temperature 100⅔°. Bowels constipated. Child developed convulsions, most marked on left side, with left arm most affected. Uttered an occasional groan. Strabismus increased to a marked degree.

Tache cérébrale was readily developed. Child quite restless. At end of

week child would not swallow. Left eye-ball became fixed, with pupil dilated. Scintillation of right eyeball marked. Convulsions more frequent and more generalized, the slightest irritation giving rise to marked general convulsions. A day later, in trying to develop the tache cérébrale, the irritation was sufficient to throw the muscles of the neck into violent clonic spasm.

Ninth day. Temperature 107$\frac{2}{5}$°.

Eye-grounds examined by Dr. de Schweinitz, and revealed an optic neuritis. The examination took place as death set in, and the changes in the retinæ were those as have been described as taking place at death.

Post-mortem, twenty-four hours after death.—There were large spots of ecchymosis over head, trunk, and extremities, most marked on left side (this took place within an hour from the time of death). On opening spinal canal and skull there was an oozing of dark venous blood.

Heart normal.

Lungs presented a few adhesions at left apex, and a very slight deposit.

The *bronchial glands* were wholly cheesy, particularly on left side. Optic nerves were not examined. The remaining organs were normal. The whole lymph system of mesentery and large intestine was swollen.

April 12, 1888.

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3. *Apoplexy.*

Exhibited by Dr. J. Leidy, Jr.

The brain substance was extensively lacerated by the hemorrhage, which was principally in the substance of the corpus callosum, corresponding to the splenium and the posterior portion of the body, greatest on the right side; the white matter of the gyrus fornicatus was involved; upon the right optic thalamus, hippocampus, and wall of the lateral ventricle were several punctiform hemorrhages. These lesions occurred in a man brought into the surgical wards of the University Hospital, in an unconscious condition, for compound fissured fracture of the left parietal bone. His chief symptoms before death were restlessness, loss of power in the left leg and arm (same side as fracture), which deepened into hemiplegia; face not affected. Temperature ranged from 99° to 103°, and toward the end the secretions were passed involuntarily. The question of trephining, the absence of hemorrhage between skull and dura, and its situation in the corpus collosum, were discussed.

September 27, 1888.

4. *Calcification of dura mater.*

Exhibited by Dr. W. Osler.

This specimen was obtained from a woman who had been for over thirty years an inmate of the out-wards of the Philadelphia Hospital, and who was admitted to the medical wards with diarrhœa.

The skull-cap showed nothing abnormal. The dura was not specially adherent, but a large portion covering the left hemisphere had undergone calcification, forming in this part a uniform plate. On the right side the process was not so advanced, but there were irregular areas, hard and dense from the deposition of lime salts. On either side of the longitudinal sinus the same condition existed; the brain was firm, the convolutions atrophied, and the amount of cerebro-spinal fluid excessive. There was senile atrophy of the viscera, but no great deposition of lime salts in the bloodvessels. *October* 11, 1888.

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5. *Pachymeningitis hæmorrhagica.*

Exhibited by Dr. W. Osler.

Specimen from a man, æt. 56 years, who died of aneurism. No symptoms of mental disease had been observed.

The skull-cap was unusually dense and thick, not specially adherent, but the number of vessels passing from it to the dura seemed greatly increased, so that the whole surface of the membrane was beset with small blood-points representing torn venules. The meningeal arteries ran in a deep groove on the inner table. The dura had a full appearance, distinctly bluish in tint, and looked as if there was a sub-dural clot. On removal the condition known as pachymeningitis was found. The inner surface presented a uniform vascular membrane, thickest over the right hemisphere, where it was at least a line and one-half in depth. The membrane could everywhere be lifted from the dura, to which it was united by loose connective tissue. It was occupied by innumerable bloodvessels, chiefly large veins. Over the vault of the hemisphere the vessels were so abundant as to give it a uniform blood-red tint. Toward the lateral sinus and the base the membrane was thinner and the arrangement of the vessels more evident. Covering the dura of the middle fossa there was a sheeting of brownish pigment,

the result of transformed blood-clot, and here the vessels seemed much less abundant. With this exception there was no hemorrhage or clot associated with the membrane. Dr. Osler remarked that this specimen illustrated (as had the two former specimens which he had shown to the Society) the early stage of pachymeningitis.

It was doubtful whether the process could be termed inflammatory. There certainly was nothing to indicate such a condition in this, or in the previous specimens. In the later stages hemorrhages occur in the membrane with the formation of laminated clots, often of considerable thickness. He spoke of the rarity of its occurrence in ordinary hospital and post-mortem work, whereas it is extremely common in lunatic asylums. In a recent paper by Wiglesworth there were forty-two cases in four hundred autopsies on lunatics. *October* 11, 1888.

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6. *Case of locomotor ataxia.*

Exhibited by Dr. H. M. Fisher.

The patient, Henry F., had been an inmate of the Episcopal Hospital for about five years. As well as could be ascertained, the disease had first manifested itself about fourteen years prior to patient's death.

During the last year of life, there had been almost complete paralysis of motion and sensation of the upper and lower extremities and of the bladder. Blindness had existed since patient's first admission to the hospital. For about two years laryngeal spasm had been an occasional symptom; during the last few months spasmodic contractions of the facial muscles had been noticed. Difficulty of deglutition was first noted a few days before the patient's death.

Sections of the spinal cord, medulla oblongata, and pons were made by Dr. W. M. Gray, of Washington, and stained by Weigert's method. He reports: "The extreme *lower* lumbar region shows a narrow band of sclerotic degeneration in the columns of Goll.

"In the middle lumbar region the degeneration embraces all of the posterior *columns*, and the posterior *cornua* are included in the lesion.

"In the *middle cervical* region the same condition is seen as in the dorsal region. In the extreme *upper cervical* region the lesion extends as a narrow band across the posterior columns, and embraces a portion of the lateral columns. The degeneration stops in the extreme lower medulla in a spot of the size of a large pin's head. The sclerosis of

these different localities is not complete, for here and there in the degenerated patches occur groups of apparently normal fibres.

"The posterior nerve-roots are completely degenerated in the *lumbar* and *dorsal* regions, but only partially so in the *cervical* region. The *anterior roots* are partially degenerated in the lower regions, but are very nearly normal in the cervical region." *November* 22, 1888.

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7. *Pachymeningitis hæmorrhagica.*

Exhibited by Drs. DE SCHWEINITZ and JAMESON.

M. S., male, white, æt. 41 years, for many years blind, admitted to the Insane Department of the Philadelphia Hospital July 9, 1887. Patient was a paretic dement, having delusions of wealth, luxuries, power, and benevolence. About six months before his death strength failed, and his happy delusional state was succeeded by a petulant, quarrelsome disposition. Occasionally he had paroxysms of anger in which he would throw himself from the bench, pound his head and limbs on the floor, and to the limit of his ability resist all efforts to protect him from his own violence. There was optic atrophy and blindness.

Three days before he died there was a left-sided convulsion which was relieved by profound anæsthesia. Twitchings of the convulsed muscles continued at intervals till death.

Autopsy, twenty-seven hours after death.—Abdominal and thoracic organs showed nothing of special interest.

Brain: Calvarium was not thickened. Dura was smooth and non-adherent to calvarium. Upon removal of the dura the right hemisphere was completely covered with a thick, flesh-colored membrane, in the posterior portion of which were numerous hemorrhages, and beneath which was a large hemorrhagic effusion. On the left side no similar flesh-like thickening of the membranes appeared, but marked opacity of the pia, everywhere with evident injection. The same thickening and opacity of the pia of the right hemisphere obtained.

In the left middle fossa there was a mass, cyst-like in structure, bearing general resemblance to degeneration of a clot. This mass was situated on the dura between it and the pia. *December* 13, 1888.

8. *Brain specimens chiefly illustrating localization.*

Exhibited by Dr. C. K. Mills.

The following specimens are from cases, diverse in character, but each having some point or points of special interest to the neuro-pathologist or neuro-anatomist. I present them with little or no comment, instead of using them separately in formal papers, hoping that the reports may prove useful to those in search of such records.

1. Softening of the Face-area with Oro-lingual Monoplegia.—C. O'L., æt. 50 years, white, salesman, was admitted to the hospital, December 15, 1888. Three or four weeks before he went to bed apparently well, but while dressing in the morning his right face and limbs became partially paralyzed; he did not fully lose consciousness, but was unable to speak.

He was carefully examined December 18, 1888, and the following record made: "He can corrugate the forehead horizontally and vertically; he can shut both eyes simultaneously; he can close the left eye alone readily, but he can scarcely close the right without the left. He cannot draw the right corner of the mouth directly upward, outward, or downward. He can easily dilate both nostrils together, but singly very poorly, though equally. He can barely manage to whistle, and in doing this he uses the left side of the mouth more firmly than the right. He has difficulty in thrusting out his tongue; the tip is usually caught behind the lower teeth, being turned down and slightly under; he can, however, manage to protrude it with difficulty, and on accomplishing this, it shows a deflection to the right of about 70°. The uvula deflects slightly to the right. He opens and shuts his mouth without difficulty."

The conclusions at this time were that the patient had distinct facial paralysis in the muscles supplied by the lower distribution of the seventh nerve; also lingual paresis; probably also a slight want of control over the right orbicularis palpebrarum. The movements, whose partial loss could clearly be determined, were those of the following muscles or muscular groups: Zygomatics, levators and depressors of the angle of the mouth and lips, platysma, orbicularis oris, and certain lingual muscles. He had some power over the nasal dilator, and good control of masseter, pterygoid, and temporal movements. The muscles of articulation were, therefore, distinctly involved, giving an oro-lingual monoparesis. He could talk, but pronounced certain words indistinctly, *e. g.*, "Columbia"—"Columna;" "Philadelphia"—"Pil-a-del-pha;" "truly rural"—"too-yooal;" "thievish"—"sibish." He could not pro-

nounce the letters "a," "j," or "w" (he called the latter "double"). He pronounced all the other letters, but had difficulty with "m" and "s." He comprehended objects and words by hearing and sight, and could read understandingly, but could not pronounce well owing to the above described articulatory defect. He had no difficulty whatever in propositionizing.

Recovery of the arm and leg seemed to be complete, but probably a little general weakness of these limbs remained.

The patient said in reference to sensation that when paralyzed the arm and leg felt as if they were "asleep"—the arm more than the leg; they both then felt cold, but this feeling had disappeared with the recovery of power. He still, however, had a feeling of numbness or of being asleep in a part of the right half of the face—most marked on the cheek below the eye. He had had pain in the foot and leg for several days after the attack, and for more than a week in his arm.

He was examined for the sensations of pain, touch, and temperature. He felt the slightest touch of the finger, or of a blunt or sharp metallic point, and could discriminate points. The tests were made most carefully both on the limbs and face. He could tell the difference between hot and cold water. He was tested on various parts of the face and forearms by Oppenheim's method of simultaneously touching symmetrical points on both sides of the body. When symmetrical points were thus touched he felt both equally, discriminating for the paretic as well as the unaffected side. Having him indicate which finger on the right hand was touched, he sometimes was correct and sometimes mentioned the finger next it toward the radial side of the hand. He seemed to make this mistake more for the ring and little finger. He made the same mistake, but not so often, on the other side. He indicated the proper finger touched, but sometimes referred the touch to a phalanx or segment of the finger near the body. Grasping the fingers at the side of the phalanx he could distinguish the motion made. (Horsley's method.)

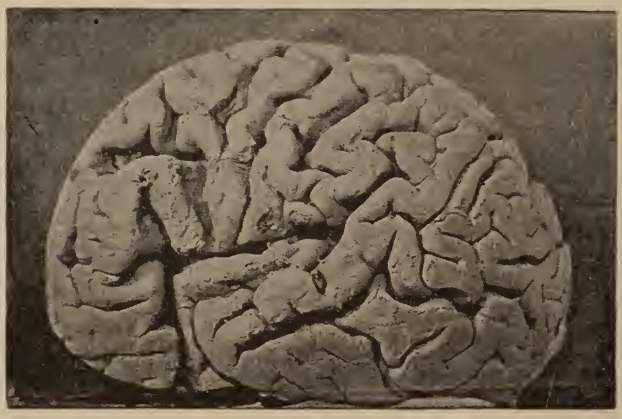
Ophthalmoscopic examination gave negative results. The veins were a little fuller than normal, the arteries natural. The disc edges were clear, and the color healthy.

He had four distinct cardiac murmurs, and all the evidences of ulcerative endocarditis, and lately of pneumonia, of which affection he died January 1st, 1889, about six weeks after he was first stricken with paralysis.

Autopsy.—Brain: A focus of strictly cortical, yellowish softening was found involving the lower extremities of the central convolutions, both on their external and Sylvian surfaces, and a spot one-half inch in

diameter about the middle of the internal portion of the island of Reil. The softening reached into the fissure of Rolando, and also into the precentral fissure, thus taking in a posterior, inferior strip of the second frontal convolution. Its greatest height was one and a half inches, upward from the Sylvian fissure, its width along this fissure one and a quarter inches. The lesion did not reach to the anterior branch of the Sylvian fissure; its anterior limit was one-fourth of an inch from this fissure. The softening was also present, as stated, on the Sylvian surface of the ascending convolutions, involving all the ascending frontal at this point, and about one-half of the ascending parietal. An embolus was felt at the bifurcation of two branches of one of the larger branches of the middle central. All parts of the brain were examined, and no other lesions were found. The focus of softening is indicated in Fig. 1.

Fig. 1.



Softening of the face-area.

Highly interesting lesions of the heart, lungs, and other organs were found, but these will not be detailed here.

Remarks.—This case presents two points of especial interest, namely: (1) The absence of propositionizing aphasia, with the presence of orolingual and facial paresis; (2) The presence of subjective and the absence of objective sensory phenomena.

The Case bears a strong resemblance to one reported by Bernheim[1] as "A Case of Lingual Mono-hemiplegia with Cortical Localization."

[1] Bernheim (Nancy). French Association for the Advancement of Science, 16th session, 1887. Gazette des Hôpitaux, p. 1016, 1887.

The patient was a girl, æt. 23 years, affected with multiple sarcomatous tumors, the first of which was observed in 1886. Suddenly (January 8, 1887), a decided deviation of the tongue supervened, the tip pointing to the right. She swallowed easily, articulated fairly well, but could not whistle. No other paralysis was present, but the pressure shown by a dynamometer was four degrees less with the right hand than with the left; three weeks later, three less. The patient died February 2d, lingual paralysis persisting. In addition to the generalized sarcomata, there was a cortical lesion consisting of an excavation of five to six mm. in depth and diameter, and caused by a sarcomatous hemorrhagic clot. This lesion was situated at the lower border of the post- and precentral convolution.

2. Intra-cerebral Hemorrhage into an Old Cyst.—A. S., æt. 56 years, black, a domestic, when admitted to the hospital was unable to speak, so that it was impossible to obtain a complete history. It was learned, however, that she had an apoplectic attack, November 15, 1888. Examination December 26, 1888, showed the lower right face paretic, the mouth being drawn to the left. The muscles affected were the zygomatic, levators of mouth and nose, depressors of the mouth, and probably the platysma. Lingual movements were uncertain; she protruded the tongue, but seemed not able to turn it upward, and moved it less freely to the right than the left. The uvula deflected a little to the right. Her mental condition was such as to make it difficult to get clear answers. She tended to repeat words spoken to her. She knew what objects were, and could hear or repeat many things correctly or nearly so, but her speech was usually much jumbled in attempting to answer questions, the difficulty seeming partly due to her impaired utterance, partly to inability to propositionize. It was not possible to test her as to reading. In the right arm palsy was absolute. In the right leg paralysis was nearly complete, as she could only extend and flex the leg slightly at the knee, and could not make any foot movement. Sensation, repeatedly tested, was normal. After taking these notes she was able to sit up until January 12th. She could not walk nor use her arm, but her speech improved a little.

January 13, 1889, she had difficulty in swallowing, and became stuporous. The next day Cheyne-Stokes breathing was noticed; and January 15th she lay motionless, and had involuntary evacuations. The left cheek and nostril puffed during respiration, but the right did not. Slight conjunctival reflex could be elicited, and was more marked during the period of ascent in the Cheyne-Stokes breathing. Her eyes were sometimes open, sometimes half shut, usually the latter during the

periods of apnœa. On calling her by name she looked as if she understood, and glanced around at times as if conscious of her surroundings, but was found not to be on close testing. She could now be stuck by a needle in either arm without response. She sank gradually.

Autopsy.—On opening the left lateral ventricle, a dark-red, almost black spot, about one-quarter inch in diameter, marked the position of a hemorrhage ready to break into the ventricle. It was one and a half inches backward from the head of the caudate body, and one and a half inches anterior to the posterior limit of the thalamus, being situated just where the tapering extremity of the caudate nucleus begins to curve around the thalamus. Transverse sections revealed a recent hemorrhage into an older hemorrhagic cyst. The anterior limit of the legion was revealed by a section three-fourths of an inch from the head of the caudate nucleus; its posterior limit at one inch from the posterior extremity of the thalamus. The greatest antero-posterior extent of the lesion was, therefore, one and a quarter inch, thinning out toward each end. The chief destruction of the ganglia and tracts was in the middle of the area thus defined. The parts destroyed in the anterior half or third of the lesion were only portions of the lenticular body and external capsule; but a section made just behind the head of the thalamus showed almost complete destruction of the lenticular nucleus, a portion of the internal capsule, and a small segment of the thalamus. A section still further back showed involvement of the lenticular nucleus and external capsule. The cerebral vessels were highly atheromatous. The entire convexity of the brain was œdematous.

3. Extensive Intra-cerebral Softening. Hemiplegia and Contracture on the Same Side as the Lesion.—E. N., æt. 44 years, was a well-known veteran at the Philadelphia Hospital, of which she had been an inmate, on and off, for twenty-five years. Nine years before her death she became a left hemiplegic, and never regained the use of the left arm or leg, although she had some little power in both. These limbs were somewhat atrophied, particularly the arm. Her mind was enfeebled. She had a mitral systolic murmur. No records of past examinations could be found, although they had been made. For several days she had been complaining of not feeling well, and October 25, 1888, while on the commode, she suddenly fell over insensible.

An examination of the patient was made a few hours later. She had evidences of an old left hemiplegia. The left upper extremity was drawn to the side, the forearm flexed at a right angle with the arm, and the hand at a right angle with the forearm, the thumb carried entirely across

the palm of the hand, the fingers usually turned inward. The contractures could be easily overcome, but on releasing them the parts returned to their contractured positions. The fingers were loose, flaccid, and easily movable to any position. They only seemed to assume a slightly flexed position from gravity or mechanical causes. They could be bent far back at the metatarso-phalangeal joints. The left leg was slightly contractured in flexion at the knee, the foot turned inward and drawn upward. The great toe was extended, pointing upward and backward, and thrown across the second toe. The other toes were bent backward at the metatarso-phalangeal articulation. The mouth was drawn to the right.

The patient lay on her back. When placed straight her head tended to the right; but her eyes at the same time had a tendency to look to the left. The pupils were nearly the same, the left a little smaller than the right. The eyes oscillated horizontally. The conjunctivæ were insensitive. The breathing was Cheyne-Stokes. When the respiration was most active it was of the puffy, tobacco-smoker's type, in part. She used the right side of her mouth in the movements of respiration more than the left. Many mucous râles could be heard. The left arm and leg were helpless, as above described; but she occasionally moved both arm and leg, especially the leg. There were slight spontaneous, as well as reflex, movements, usually of flexion. Handling the left arm or leg sometimes set up a clonus or trembling of the limb. Her general condition was such that it was difficult to decide as to the paralysis of the right side of the body. The right limbs certainly did not present any contractures. It was also difficult to make any certain tests for sensation, but the limbs responded by movements to prickings on the right side better than the left. Knee-jerk was present on both sides, perhaps increased on the left. Every few minutes she had what appeared to be a left unilateral spasmodic seizure, in which the left leg was drawn up and moved at the thigh and knee; the left upper extremity had a lifting-upward and outward movement from the shoulder, and the face was distinctly drawn up to the left. Sometimes the arm alone moved. While studying her it was noticed that she very seldom moved her right arm or leg. Such movements as were seen, whether volitional or not, were usually on the left side.

The heart-sounds were irregular, tumultuous; no differentiation between the first and second could be heard; a mitral murmur could be made out, but its character could not be determined. Her urine, drawn by catheter, was cloudy, pale; specific gravity, 1026; reaction, acid;

sediment white and large; pus in considerable quantity; a small amount of albumin.

Autopsy.—No lesions were found in the brain cortex, with the exception of an ecchymosis of the pia-arachnoid over the upper third of the fissure of Rolando of the right hemisphere. Nothing was found in the ventricles. Softening was present of the middle portion of the lenticular nucleus and internal capsule, and of the island of Reil of the left hemisphere; the white matter of the occipital lobe, and of the posterior half of the temporal lobe of the left hemisphere, was also softened and broken down. The softening appeared to be due to blocking of the cortical, temporal, and some ganglionic branches of the middle cerebral, and also of some branches of the posterior cerebral arteries. Old mitral and aortic disease and pathological conditions in various organs were present.

4. Old Hemorrhagic Cyst, with Aphasia and Hemiplegia.—W. M., æt. 65 years, white, was admitted to the hospital in February, 1887. It was impossible to obtain any information from the patient on account of his aphasia and mental condition. It was noted on admission that he was a right hemiplegic, with contractures and aphasia almost complete, the only words he was able to say being "yes" and "no," usually accompanied by gestures. He had an ulcer on the right leg and one on the knee. His urine dribbled continually. His heart was hypertrophied; the vessels were atheromatous. His urine contained bile and coloring matter, but no albumin or sugar. Finger clonus of the left hand and ankle clonus were present.

Notes of an examination were made January 19, 1889. Inspection showed some drooping of the right face. He was apparently unable to thrust out his tongue, and said, "Oh, my!" when asked to do it. He was not only unable to put out his tongue, but had little use of it in his mouth. The uvula was straight and the palate not very responsive. His tongue had a tendency to bunch at the back of his mouth. As nearly as could be determined, therefore, he had facial paresis in the lower distribution of the nerve, and lingual palsy.

The right arm was completely palsied; he could not move the fingers or any part of the arm. The arm was carried to the side, and the forearm usually at right angles across the trunk; the hand was contractured, the middle, ring, and little fingers being bent inward at the metacarpophalangeal articulation; the thumb flexed, adducted, and thrust between the index and the middle fingers, and the index finger flexed over the thumb. The upper extremity was not permanently rigid at the shoulder, elbow, wrist, metacarpal and phalangeal articulations; *i. e.*, it could

be straightened by force, again assuming the old position. It measured an inch less than the left around the biceps. He was apparently unable to move the right leg and kept it in one position. The thigh was strongly flexed on the pelvis, and the leg on the thigh at more than a right angle. The leg could not be straightened, as could the contractured arm. The four outer toes were very slightly turned downward. The ankle-joint was free. The foot was carried in a normal position, except the toes. The left leg apparently was not used and had a tendency to contracture, like the right.

A marked condition of dark bronzing was present from the knees down both legs, except the soles of the feet. The arms, particularly their outer aspects, showed the same tendency in spots, but not so extreme. Below the knees it was uniform, as if the limbs were those of another man of a different race. He evidently had sensation to slight touches of sharp points, his face and manner indicating pain.

The brain lesion can be best described by the simple statement that an old cyst of immense size was present, involving and destroying the island of Reil and the anterior two-thirds or more of the capsules and the two striate bodies.

5. Tumor of the Dura Mater.—J. J., æt. 32 years, white, born in Scotland, laborer, was admitted to the hospital August 9, 1888, complaining of severe headache and sleeplessness, which had existed for three months. He was obstinately constipated and had lost appetite and flesh, although he was still a large, muscular man. His breath was offensive; his urine was normal. Knee-jerk was normal. No paralytic symptoms were present. The right pupil was rather larger and responded to light more slowly than the left. Eight years before, while serving as a policeman in Scotland, he had been struck on the back of the head with a black-jack, was rendered unconscious, and remained so for a considerable time, and after this he suffered considerably with headache, but finally recovered. He attributed his attack to this accident and the abuse of alcohol. He denied syphilis, but admitted having gonorrhœa fourteen years before admission. At the time of his last attack he was working for a plumber in different parts of the city, until his headache became so severe that he had to quit. He located the pain over the right eye. It varied in intensity, always being worse at night.

On admission his temperature was 100°; pulse, 50; and respiration, 18. The next morning his temperature was 97°; pulse, 48; respiration, 18. He was extremely restless, tossing about, but perfectly conscious. His urine was scanty and high-colored, and passed only at long intervals. He was quieter and somewhat dull the third morning, and the

dulness increased, so that by 10 A.M. he had to be aroused to answer questions, and then answered slowly but intelligently. Two hours later he was completely comatose with labored respiration. At 1.30 P.M. a decided change was noticed; his breathing became stertorous and slow; his pulse, which had been above 48, increased to 130; respirations dropped to 10, then to 6, and after two or three ineffectual efforts at inspiration, breathing stopped. The pulse continued to beat for fully five minutes after the cessation of the respiration. Artificial respiration was tried with no effect.

The *autopsy* showed thickening of the skull about the size of a silver dollar, beginning one inch above the line of the external meatus; the skull at this point was strongly adherent to a tumor or thickening of the dura mater. The mass was flat, almost circular, one-eighth of an inch in thickness, tough, and whitish-yellow in appearance. The dura and pia mater were agglutinated and adherent to the brain beneath the growth, and both skull and brain were bruised and eroded. The growth was so situated as to cross the fissure of Sylvius over portions of the posterior central, inferior parietal, and first temporal convolutions, its anterior limit being the middle of the latter. The lesion was strictly meningeal, but in enlarging had slightly invaded both the skull and brain substance. Subsequent examination showed the growth to be syphilitic.

6. TUMOR OF THE ANTERIOR FOSSÆ AND ORBITAL LOBES.—M. K., æt. 72 years, was admitted to the hospital unconscious with a history of epilepsy, or at least of convulsions. When admitted her breathing was puffing and she was in a stupor. Albumin was found in her urine, and she came out of the stupor after a hot-air bath, and improved under digitalis and ammonia, but she remained very dull, and her answers to questions were usually neither relative nor coherent. Her mouth was drawn a little to the left, and the right arm was paretic. Sensation was preserved. The skin reflexes were marked. She again sank into a stupor and died.

At the *autopsy* the dura mater was found to be strongly attached to the skull and to the pia mater over the postero parietal region. Scattered ecchymotic areas were found, particularly over the left hemisphere. The pia stripped readily from the brain. Some of the convolutions were somewhat atrophied. Superficial softening of each caudate nucleus was found—an irregular patch about the size of a dime, extending only a few millimetres into the brain substance. A large, soft, purplish tumor filled up the floor of the skull and the longitudinal fissure between the anterior extremities of each hemisphere; the growth reached from the optic

chiasm to the tips of the hemisphere, invading the orbital lobes and involving the olfactory bulbs. Close examination showed that it chiefly encroached upon the left hemisphere, the convolutions invaded being the mesial and orbital surfaces of the first frontal and the extreme anterior portion of the gyrus fornicatus.

7. Specimen Showing the Relation between the Retro-insular and Superior Temporal Convolutions.—Within the Sylvian fossa, behind the island of Reil, two or three convolutions usually present have received but little attention from students of the brain. These are known as the retro-insular or temporo-parietal convolutions.

Fig. 2.

Relation of the first temporal to the retro-insular convolutions.

A glance at them, particularly as seen in brains in which the operculum is imperfectly developed, shows that they are connecting gyres between portions of the temporal and parietal lobes, as the convolutions of the insula are apparently between more anterior parts of the temporal and both the parietal and frontal lobes. In a number of human brains I have observed indentation or cross-fissuration of the first temporal convolution, indicating a tendency to confluence between the anterior portion of the first temporal fissure and the fissure which separates the two most anterior retro-insular convolutions.

The specimen showed almost complete confluence between these fissures, making the anterior half of the first temporal continuous with the first retro-insular convolution, the other retro-insular gyres blending with the posterior portion of the first temporal.

This specimen and the others referred to make it probable that the first temporal and retro-insular convolutions hold morphologically and functionally some close and peculiar relation.

8. The Brain of an Idiot.—The patient was a boy, æt. 10 years, who had been idiotic from birth. He was completely aphasic, and was paralyzed, with contractures on both sides of the body, but much more markedly on the right.

Fig. 3.

Left hemisphere of the brain of an idiot.

A small portion of the cerebellum was uncovered by the cerebrum. The island of Reil was uncovered in both hemispheres. On the left side, owing to the great arrest of development of the third frontal, central, and inferior parietal convolutions, the insula and retro-insular convolutions were seen in their full extent, the gap between the fronto-parietal and temporal lobes being at its widest point one and a half inches.

The specimen affords a good opportunity of studying the relations of the insula and retro-insular convolutions to the temporal, parietal, and frontal lobes.

The insula and retro-insular convolutions were clearly distinct from each other, the latter, three in number, connecting the posterior half of the first temporal with the inferior parietal convolution. The anterior half of the first temporal was more closely related to the convolutions of the island, and these in their turn with the central and frontal convolutions, particularly the third frontal.

The island of Reil and retro-insular convolutions of the right side were also clearly exposed, the gap, however, at its greatest width measuring only one-half an inch. In both hemispheres the fissures and convolutions were generally simple and low in type and arrangement. On both sides, for example, the first and second frontal, the pre-central, central, retro-central, and interparietal fissures were well defined and easily determined, and the fourth and fifth temporal fissures demarcated typical lower temporal convolutions. In the parieto-occipital region the gyral and fissural arrangements were ape-like, showing peculiarities of development in the transition of gyres. The left first temporal fissure almost communicated with the parieto-occipital and the posterior arm of the interparietal fissures. The left first occipital or superior parieto-occipital annectant convolution was ill-developed and just below the surface. On the right the anterior arm of this transition convolution of Gratiolet was depressed, but the posterior was well to the surface. On both sides the parieto-occipital and calcarine fissures were separated by an inferior internal connecting convolution, larger on the left. The cuneus on each side was fairly well-developed and of good size for the size of the brain, the only apparent arrest being of the portion formed by the posterior arm of the first occipital convolution. On both sides the calloso-marginal fissures were badly defined—on the left separated into two parts by a large convolution, on the right separated into three parts by interposed convolutions.

9. Occipital Lobes of a Man Blind more than Twenty-five Years.—The patient from whom these specimens were obtained died at the Philadelphia Hospital, alleged to be between seventy and eighty years old. He was a harmless dement, positively known to have been blind for more than twenty-five years.

The notes will be confined to a brief description of the occipital lobes and adjacent parieto-temporal region (the portions of the brain included by different observers in the visual area).

Left hemisphere.—The cuneus was small. The arrest of development was apparently more on the side of the calcarine fissure. This fissure was crossed by a convolution in its posterior third. The parieto-occipital, calcarine, and hippocampal fissures showed a decided tendency to confluence. The first occipital convolution of Ecker (superior external *pli de passage* of Gratiolet and par-occipital of Wilder) showed decided atrophy or lack of development; its posterior arm was a much-narrowed convolution, so depressed below the surface as to allow partial confluence between the parieto-occipital fissure and the interparietal. The entire occipital lobe was unquestionably small; the second and third

occipital convolutions of Ecker were readily made out, but presented a narrow, dwindled appearance. The parallel fissure was imperfectly developed, bridged in its posterior third, and turned upward just back of the Sylvian fissure. The angular gyre was fairly well developed, occupying a position just in front of the line of the parieto-occipital fissure, which was apparently thrust back.

Right hemisphere.—The cuneus was small. The calcarine fissure was imperfectly bridged near its posterior extremity. The same tendency to confluence of the parieto-occipital, calcarine, and hippocampal fissures, and the absence of internal annectant gyres were noticeable here, as in the other hemisphere. Here also, as upon the other side, the first occipital fissure of Ecker was small and depressed below the surface. The entire occipital lobe posterior to the parieto-occipital fissure was again unquestionably small. The conditions about the parieto-occipital fissure in both hemispheres were somewhat ape-like and like those which have been found in the brains of paranoiacs, criminals, negroes, and idiots. The first temporal or parallel fissure was well marked in this hemisphere, the angular gyre being entirely in the parietal lobe and of fair development.

10. Brain of a Man, Deaf-mute more than Thirty Years.[1]—It is only necessary for the purposes of this report to state that the patient from whom this brain was removed was an old man who had been deaf for more than thirty years, and had no disease of the brain. A photographic view of the left hemisphere is given in Fig. 4.

The first temporal convolution of the left hemisphere was *narrow and very smooth*, particularly in its anterior half and posterior fourth, while about the centre of the convolution was a secondary cross-fissure which nearly divided it into two parts, this fissure being continuous with that separating the two retro-insular convolutions. At its posterior extremity the first temporal convolution was either split longitudinally into two parts, or else the lower of these might be regarded as the posterior extremity of the second temporal. As a whole, the first temporal convolution appeared less in bulk than usual. The parallel fissure was not quite continuous. At about the junction of its fifth with its last sixth it was bridged below its surface by a small connecting convolution. This fissure turned up close behind the horizontal Sylvian. The second temporal convolution showed no very distinct antero-posterior fissuration separating it from the third convolution. Posteriorly a fissure ran antero-posteriorly for a distance of two inches, and if this was regarded as the

[1] Presented by Drs. Charles K. Mills and Roland G. Curtin.

inferior boundary of the second temporal convolution, the latter was very small. The middle third of the second and third convolutions were fused, either together or the middle portion of the second was lacking. The fourth temporal or fusiform, the lingual, hippocampal, and uncinate gyres were well defined. The left third frontal gyre was small, particularly in its hinder part, the posterior portion of the arch around the ascending Sylvian being very narrow. The lower extremities of the central convolution were narrow and pointed; the band of convolution between the Rolandic and Sylvian fissures very thin. A small segment

FIG. 4.

Left hemisphere of the brain of a man, deaf-mute for more than thirty years.

of the island of Reil could be seen when the lobes bordering the fissure of Sylvius were drawn close together. The island appeared to be diminished in size.

On the right side the first temporal convolution was rather smaller than usual, but did not present marked diminution shown by the first temporal on the other side; it was smaller in its anterior half. The parallel fissure turned upward further back than on the left side. The antero-posterior separation between the second and third temporal convolutions was better, so that the two convolutions could be defined. The other temporal convolutions were of fair size and definition. The right

posterior portion of the third frontal was atrophied. Comparing the right with the left, the lower extremities of the two central convolutions on the right were not so pointed or dwindled as on the left. The loss of bulk on the right seemed to be more confined to the posterior portion of the third frontal. Both occipital lobes were well developed, a marked difference in this respect being shown between this brain and that of the blind person.[1]

Making a comparative general statement, it may be said that the occipital lobes of this deaf-mute brain were distinctly larger, and the upper temporal convolutions distinctly smaller than in the brain of the blind man. Both hemispheres of this brain showed the close connection between the retro-insular and the anterior portion of the first temporal, and the posterior retro-insular with the posterior part of the first temporal convolution. The motor and pre-frontal lobes, with the exception of the third frontal as detailed, were well developed. The acoustic striæ were evidently atrophied. They were compared with two other specimens and were found to be greatly attenuated.

January 24, 1889.

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9. *Asymmetry of the olivary bodies of the medulla oblongata.*

Exhibited by Dr. A. V. Meigs.

A little more than a year ago I was able to exhibit to the members of the Society the medulla oblongata of a baby, three months of age, that died of infantile atrophy, which presented a great difference in size of the olivary bodies. I now present for your examination an accurately made drawing, and photographs, and sections of the medulla itself. These were prepared for me by Dr. William M. Gray, of the Army Medical Museum in Washington. Examination of the sections fails to show any abnormality in the microscopic structure, though, of course, the smaller olivary body presented upon its external surface a much less full and rounded curve than the other one, and the *nucleus dentatus* is seen to be smaller, and apparently less complicated in its windings. Last year I mentioned that I had seen and made sections of the medulla of a man 51 years of age, who died of Bright's disease, whose left olivary body was much smaller than the right. In this case there was also

[1] I am indebted for the photographs with which these reports are illustrated to the courtesy of Dr. B. Alexander Randall, of Philadelphia.

quite marked asymmetry of the two sides of the medulla, which at the time I supposed to be due to some imperfection of the methods of preservation and hardening, and preparation for microscopic examination; but now I am quite satisfied of the contrary, as the same asymmetry was perceptible, though in less degree, in the case of the child above mentioned. During the last year I have had occasion to make quite a number of post-mortem examinations, and in some instances have been able to investigate the condition of the olivary bodies. Of infants I have records of five cases in which a note was made of their condition, and in two out of the five (including the one first mentioned) they were unsymmetrical. These cases were as follows: The one first mentioned, a male,

three months old, died of infantile atrophy (marasmus), and the left olivary was the smaller; a male infant, three months old, died of acute hydrocephalus, and the left olivary body was the smaller; a male infant, five weeks old, died of whooping-cough, and the olivary bodies were symmetrical; a male, six months of age, died of infantile atrophy, with various complications, and the olivary bodies were symmetrical; a female infant, six weeks old, died apparently of kidney disease, and the olivary bodies were symmetrical.

I am sorry I have nothing more complete to record in regard to this interesting anomaly in adults, but I am almost sure that I found the condition of asymmetry present once last summer in making an autopsy at the Pennsylvania Hospital, though I have not succeeded in laying

my hands upon the notes of the case. The sections of the medulla of the infant in which the anomaly was first noticed show that the left side generally is perhaps a little smaller than the right, the condition not being confined to the olivary body alone, and this was more marked in the man who died of Bright's disease, as has been already mentioned. It is curious that in each of the three cases it should have been the *left* olivary body which was the smaller; and it is worthy of note, too, that they were all males. The difference in size of the two bodies is evidently due to imperfect development of the smaller one, and not to overgrowth of the larger.

So far as I am aware, this condition of asymmetry of the olivary bodies has not been previously noticed, though I confess that I have not made a very extensive examination of anatomical literature.

It is an interesting question whether the condition has any pathological bearing, and it has been my intention to examine carefully the medulla oblongata in cases of Bright's disease, but no sufficiently typical case has come under my notice as yet in which I was able to do so. At the present time, therefore, it is only possible to place my observations on record as an anatomical anomaly which is either very rare or has been overlooked. *February* 14, 1889.

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10. *Specimen from a history of a case of abscess of brain.*

Exhibited by Dr. F. A. Packard.

The patient from whom this specimen was obtained was admitted into Dr. Ashhurst's ward at the Pennsylvania Hospital at 10.30 A.M., February 23, 1888. On admission she was absolutely comatose, with complete right-sided hemiplegia, inequality of the pupils, the left being dilated, absence of conjunctival and retinal reflexes, slow pulse, stertorous breathing, and blue lips. From the left ear there was a discharge of fetid pus, and in the external auditory meatus was a small granulation polyp.

The history of the case, as given by her attending physician, was that in the early part of February she noticed discharge from the left ear, and suffered from severe left-sided headache. On February 18th her speech became slightly thick, this condition rapidly progressing until on February 20th there was complete aphasia without loss of word appreciation. At 10 P.M. on the night before her admission to the hospital complete coma with right-sided hemiplegia set in.

In about three-quarters of an hour after her admission Dr. Ashhurst trephined. An incision of a semicircular shape was made a little in advance of and above the left external ear. A large trephine was used, and the opening enlarged by means of Hopkins's forceps. The brain, covered by opaque dura mater, immediately bulged through the opening. There was no visible pulsation, either at that time or shortly after, when an incision was made through the dura mater, exposing the brain substance, which was of a dirty-yellow color, soft, and without any appearance of gray matter. A director was then inserted downward and backward without meeting any pus, although the soft, broken-down brain substance almost flowed along its groove. A drainage-tube was inserted along the track made by the director, and a light $HgCl_2$ gauze and cotton dressing applied.

After the operation she moved her right arm a little when tickled in the ribs, the pupils evened up, and her color improved with the improvement in respiration and circulation. Toward evening she could swallow, but remained unconscious and hemiplegic, although the reflex motions of the right arm excited by tickling persisted, as did also conjunctival reflex. Slight left-sided clonic spasms appeared at intervals in the arm and leg. On the next day her improved condition still held until evening, when the respirations again became stertorous, coma deepened, and the circulation became feeble. Her temperature at 3 P.M. suddenly shot up from 99⅘° F. to 104° F., and in spite of acetanilide and cold affusions steadily rose to 105° F. She died at 1 A.M., February 25, 1888.

Autopsy, held nine hours after death.—Brain only examined. Rigor mortis equally pronounced on the two sides. Dura mater slightly adherent to calvarium. Longitudinal sinus contained a small amount of clotted blood. Dura mater thickened about petrous portion of left temporal bone. The left temporo-sphenoidal was distended, soft, and fluctuating. In removing the brain an abscess cavity burst in that lobe and discharged about two fluidrachms of fetid greenish pus before the opening could be closed. On the under surface of this lobe was a greenish area corresponding in size, shape and position to a necrotic area later found in the petrous bone. The whole temporo-sphenoidal lobe was converted into a thin-walled abscess cavity. The pia-arachnoid was adherent over the whole lobe. In the left lateral ventricle were a few drops of yellowish pus, and the ependyma was rough and opaque, with bands of lymph uniting the two surfaces. The basal nuclei on the left side (chiefly the corpus striatum) were soft and yellowish. Right side of the brain was normal.

On removal of the brain and dura the petrous portion of the left tem-

poral bone was seen to present on its posterior surface about one quarter inch to outer side of internal auditory meatus, a ragged necrosis opening one-third inch in diameter. Into this opening a probe passed, and emerged easily out of external auditory meatus.

February 28, 1889.

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11. *Specimens from a case of cerebral abscess secondary to ear disease.*

Exhibited by Dr. B. A. Randall.

I desire to present the specimens from a case of abscess of the cerebrum consequent upon suppuration of the tympanum and caries of the temporal bone. hoping that they will have for others some of the interest they have for me.

Maria W., æt. 4 years, of Russian-Polish birth, though doubtless, from her name, of German stock, was admitted to the Children's Hospital on August 22, 1888, suffering from suppuration of the mastoids and with strumous suppuration of the left elbow-joint and thumb. Facial palsy was complete on the left side, with considerable fetid discharge from both auditory canals and from a small cicatrizing sinus behind each auricle. No history could be obtained, except that the condition had been for some time present, and that the mother had died after a lingering illness. When I first saw her, early in October, both temporal bones showed marked caries; the auditory meatus on each side was nearly filled with polypoid granulations, more marked and obstructive on the left, and dead bone could be felt in all directions by a probe introduced through either meatus or post-auricular sinus. Successive crops of polyps were removed and the ears cleansed by free syringing, followed by insufflations of boric acid; and a number of sequestra, some as large as could be extracted through the auditory canal, were from time to time removed from each side. These sequestra were generally black and friable, and their exact origin was not recognizable. The discharge soon decreased in quantity and offensiveness; granulation was slightly redundant only on the left, and although the loss of substance evidently involved the labyrinth on the left, and seemed almost as great on the right, the child undoubtedly had some hearing and seemed in a fair way to recover. The suppuration from the elbow and the thumb had continued profuse and exhausting, and was now evidently the cause of the hectic fever shown by the temperature charts; so on November 21st Dr. Ashhurst amputated the thumb and excised the elbow-joint. The

operations were fairly well borne and the healing progressed pretty well; but the child gradually lost ground, and growing rapidly worse during the last days of the year, died on the evening of December 31st. A large quantity of pus escaped from the left ear shortly before death, although the regular syringing had brought away very little for weeks before. No paralysis except that of the facial was at any time noted; and when, about the middle of December, I undertook an ophthalmoscopic examination the unremitting struggles of the child and the rolling of the eyes completely foiled me, in spite of fully dilated pupils and a perseverance as long as seemed at all justifiable, in view of the child's weakness. The left cornea was opaque near its lower margin by reason of the imperfect protection and lubrication afforded by the paralyzed lids, and there was some other lack of transparency of the media; but I fail to understand the difficulty experienced in obtaining any real view of the eye-grounds. The orbital contents were removed at the autopsy and no distention of the nerve-sheaths was found. There seemed, however, some swelling of the nasal margin of the right optic disc. The left eye has not been opened.

Autopsy was made, twenty-four hours after death, by Dr. Eckman, resident physician of the Children's Hospital, on January 1st, in my absence. The body was much emaciated. The meninges appeared normal except over the left tegmen tympani, where the dura was destroyed of an area 10 mm. in diameter, and an irregular gap was found in the bone beneath it. An organized clot in the outer part of the left lateral sinus was loosely attached to the sinus wall, which seemed normal, and no involvement of the underlying bone was found.

The convexity of the brain seemed normal, and there was little or no exudation at the base. A large abscess in the left spheno-temporal lobe above the opening in the roof of the typanum had evidently burst, and evacuating itself through the ear, had given rise to the gush of pus which preceded death. Further laceration of the softened cerebrum took place in removing it, and a communication between the abscess cavity and the lateral ventricle was found which probably did not preëxist, as there was little pus in the left ventricle and none in the right. The left internal auditory meatus was full of pus and no trace of the seventh or eighth nerve can now be found. They may possibly have been accidentally destroyed. Both nerves seemed normal on the right. The right hemisphere, the cerebellum, and the medulla were normal.

The lungs were full of cheesy nodules, more numerous on the left, where some of them had formed cavities as large as a big pea in the apex and elsewhere.

The heart contained a chicken-fat clot in the right ventricle, but was otherwise normal, as were the kidneys and spleen and other organs examined.

It seems interesting to contrast this case with one which Dr. de Schweinitz presented for me, where the carious process had destroyed the tegmen tympani and the wall of the lateral sinus, but the dura had been thickened by a pachymeningitis which effectually shut out infection through these channels, while pus entering through the internal auditory meatus had set up a fatal basillar meningitis.

February 28, 1889.

VII. ORGANS OF THE SPECIAL SENSES.

1. *Gun-cap injury of the right eye; retention of the cap; sympathetic irritation; enucleation on the twentieth day.*

Exhibited by Dr. G. E. de Schweinitz, for Dr. S. D. Risley.

J. S., æt. 17 years, who presented himself for treatment October 8th, had more than three weeks before received an injury of the right eye from the premature explosion of a cartridge. The eye was immediately blind, and there was profuse bleeding. The accident was followed by twenty-four hours of severe pain, since which he has been free from suffering, except for dread of light in the left eye. When first seen (October 8th) the active inflammatory reaction had subsided. The ball was small, the sclerotic of a muddy-yellow color, and there was a depressed and puckered scar near the inner corneal limbus with a small button of granulation sprouting from the centre. There was a large coloboma of the iris at the nasal side, its base corresponding to the scleral scar. The lens was semi-opaque, and of a straw-yellow color. With strong oblique illumination a large yellowish white mass could be seen occupying the ciliary region at the site of the scar, the mass being readily studied through the broad gap in the iris. The ball was extremely tender. The friends of the young man thought there was no part of the cartridge-case in the eye, as they believed they had found every part of it. Enucleation was performed on the 11th. The entire cap of the cartridge was found deeply imbedded in the vitreous chamber, having carried before it the iris, ciliary body and processes, and was apparently wrapped upon them. *October* 13, 1887.

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2. *Ulceration of the cornea; prolapse of the iris, with escape of vitreous, the result of purulent ophthalmia; enucleation.*

Exhibited by Dr. G. E. de Schweinitz.

Maggie S., æt. 35 years, was attacked with double muco-purulent catarrh. This was treated with various domestic abominations, and

steadily grew worse. When admitted to the Philadelphia Hospital the left eye was ruptured, the iris and vitreous protruding, the ball shrunken, and intensely tender. In the left eye, in addition to the ophthalmia, there was a large crescentic ulcer of the cornea. The woman was suffering most keenly, her temperature elevated, her intellect slightly clouded. The left eye was removed. The pain disappeared, the temperature became normal on the following day, and the healing was natural. Through the large perforation the contents of the globe behind could be seen protruding. In spite of an existing panophthalmitis, which has been held to contra-indicate enucleation, the operation in this case was followed by the happiest results. *October* 13, 1887.

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3. *Shrunken eyeball; complete calcification of the lens.*

Exhibited by Dr. G. E. de Schweinitz.

Annie H., æt. about 24 years, an inmate of the Eye Wards of the Philadelphia Hospital. She has for years been a sufferer from granular lids. The right eye shows well-marked granulations upon the lids; pannus at the upper part of the cornea and trichiasis. The left ball was a small shrunken body deeply imbedded in the socket, and almost covered by the thickened and granular conjunctiva. This eyeball became very painful, and the woman began to have severe headaches. Distinct tenderness over the upper ciliary region of the right ball was also manifest. Enucleation of the shrunken eye was performed, and incision into it revealed a completely calcified lens. *October* 13, 1887.

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4. *Eyeball showing deep lacerated wound of the ciliary region from injury by an oyster-shell.*

Exhibited by Dr. G. E. de Schweinitz.

L. K., æt. 9 years, on his road home from school engaged in some rough play. He was struck in the left eye with an oyster-shell. The upper lid was wounded, and a large transverse cut in the cornea extended out one-quarter of an inch into the sclera. Through the gap a prolapse of the iris and ciliary body protruded, and at the outer angle

vitreous was presenting. The eyeball was shrunken, the boy pale and shocked. Enucleation was performed on the following day. No foreign body was found in the eye, but the deeper tissues were disorganized from the effects of the blow. *October* 13, 1887.

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5. *Sarcoma of the conjunctiva.*

Exhibited by Dr. G. E. de Schweinitz.

This specimen consisted of one larger and several smaller pigmented masses, which I removed from the corneo-scleral margin of the left eye of a man æt. 36 years. The history of the growth was briefly the following: Four years ago a small black speck appeared near the corneal margin, and gradually expanded until it formed a growth about the size of a large pea. This was removed in the spring of 1885. About a year later it began to recur, and grew until December 7, 1888, at which time it presented the following appearances, viz.: an elevated mass about the size of a hickory nut, partly pigmented, and firmly attached to the corneo-scleral junction. From this, the surrounding conjunctiva spread out into thickened layers far back into both upper and lower retro-tarsal folds. This conjunctiva was deeply pigmented, and a few spots of similar pigment were apparent on the tarsal conjunctiva. *March* 8, 1888.

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6. *Dermoid Tumor of the Cornea.*

Exhibited by Dr. G. E. de Schweinitz.

This tumor was found in the left eye of a male child born two weeks ago in the Philadelphia Hospital. The growth was present at birth. It consisted of a globular swelling, one-half a centimetre in breadth, situated partly upon the cornea and partly upon the sclera at the lower and outer portion of the eyeball. The walls of the tumor were dense and resisting, pinkish in color at the base, yellowish-white on the top, where the surface was stippled in appearance, and from which a number of light-colored hairs protruded, each a few millimetres in length. A little sebaceous matter covered the surface. No other congenital malformations of the eye or its appendages existed in this child, and in its

general state it was a perfectly normal baby. As is well known microscopical examination of these growths may reveal structures resembling the skin and its accessories, fat, connective tissue, hairs and hair follicles, or simply dense fibrous tissue. Exceptioually the development of hair from these tumors is remarkable. In Wardrop's well-known case twelve long hairs grew from the middle of such a cyst, passed between the fissure of the lids and hung down upon the cheek. Occasionally these dermoid cysts are double. Dr. Taliaferro, of Kentucky (*American Journal of the Medical Sciences*, 1841), has recorded a good example of this kind. The tumors arose from the lower part of each cornea. The larger, which was cone-shaped, grew on the left eye, and on its apex was a number of long hairs. Dermoid cysts are not uncommon at the outer part of the eyebrow, where they may be beneath the orbicularis and may invade the subjacent bone. Less frequently they occur above at the inner angle, and may be mistaken for a meningocele. The solid dermoid tumors of the eyeball, such as the example presented this evening, are more unusual. According as they are situated entirely upon the conjunctiva, and are movable, or partly upon the cornea, and are fixed, they are classified by systematic writers as dermoid tumors of the conjunctiva, or dermoid tumors of the cornea. When their position is the latter one they cannot be entirely removed and on dissection it will be found that they may penetrate deeply into he corneal tissue. *March* 8, 1888.

VIII. TUMORS NOT OTHERWISE CLASSIFIED.

1. *Mediastinal tumor.*

Exhibited by Dr. T. S. Westcott.

A. D. R., æt. 28 years, brakesman, born and residing in Harrisburg, admitted to the University Hospital September 5th, died September 21st. Was a man of good personal habits; no venereal disease; no injury about the chest, but had had his hand crushed between the bumpers of freight cars, from which he recovered with a deformed and almost useless member, several years before. According to his own story, he had been perfectly well until about June 1st of this year, when he began to suffer from dyspnœa and took to the house. At this time, too, he noticed the presence of a soft tumor above the clavicle of the left side which had been increasing in size. The voice had apparently not been altered in quality, but at times he had a short, sharp, bark-like cough, attended with expectoration of viscid mucus. There was no inequality in the size of the pupils. He took solid food without difficulty, and complained of no pain or discomfort except from his orthopnœa and consequent loss of rest. The circulation was good; no œdema of arms or face, and no difference in pulsation of the right and left radials were observed.

Physical examination showed a firm, lobulated tumor on the left side of the neck springing from beneath the posterior border of the sterno-mastoid, evidently an involvement of the deep cervical lymphatic glands. The mass could be traced downward beneath the clavicle and over the whole anterior aspect of the left chest, reaching from the right border of the sternum, masking the heart, the pulsation of which could be heard very faintly at the sixth interspace, at left edge of sternum. From the anterior axillary line around to the vertebral column absolute flatness was elicited, with absence of respiratory sounds, but with a sense of elasticity to touch not observed over the anterior portion of chest. Hypodermic puncture in this area revealed the presence of dark purulent liquid. The axillary glands were also greatly enlarged upon the left side.

The post-mortem showed a solid, pinkish, fleshy tumor, in some portions extremely hard and almost cartilaginous, filling the entire mediastinal spaces, enveloping the trachea, the aorta, and all the great vessels

of the neck, reaching from the root of the neck almost to the diaphragmatic attachment, and from the right border of sternum to the left anterior axillary line. The apex of the left lung had been pushed downward and posteriorly and the rest of the viscus was collapsed. Fluid filled the cavity of the pleura. The growth had firm fibrous attachment to the bodies of the first four or five dorsal vertebræ, and had probably started here in the posterior mediastinal glands. The pericardium was partly hidden under the anterior lower edge of the tumor. By a continuation piercing the first intercostal space the growth was continuous with the axillary and cervical tumors. The bronchial glands were involved and a few nodules appeared on the surface of the lungs themselves near the roots. The mesenteric glands were likewise greatly enlarged. The heart was small but not diseased, and the other organs were healthy. The œsophagus was of normal calibre and had been simply pushed over to the right, which accounts for the absence of dysphagia. The tumor, with the heart and vessels, weighed nearly seven pounds. A microscopic section taken from the oldest portion of the growth showed an almost entirely fibrous structure. Sufficient time has not yet been offered to make a careful examination; but it is altogether likely that it is a fibro-sarcoma.

The most remarkable aspect of the case is the fact that the pressure symptoms, save those of the respiratory function, were so incommensurate with the enormous neoplastic development. *September* 22, 1887.

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2. *Sarcoma of the skin.*

Exhibited by Dr. J. H. Musser.

The enormous mass which you see here, the surface of which is irregularly ulcerated, representing the skin of the scrotum with invasion of the skin of abdominal walls and pubes and the tissue in the groin, was removed from a patient after death. The account of the state of the internal organs I regret to be unable to furnish, as no autopsy was allowed. In addition to this lesion which I present, a mass, during life, was seen at the lower border of the manubrium, growing from the anterior mediastinum. The skin over the mass was not affected. It was the size of a large fist, very firm, and seemed to have absorbed the sternum in its growth outward. A flat, firm, dark-red mass, two inches in length and one in width, ovoid in shape, was found on the forehead above the left eye. Sections from the skin show the disease to be due to sarcoma.

The patient, A. M., æt. 34 years, single, a laborer, of bad habits, was admitted on August 7th of the present year. He had been in the Pennsylvania Hospital and also in St. Agnes' Hospital for the present ailment. He has had no previous illness, save typhoid fever eight years ago. Last January he had an attack of gonorrhœa, and he states that thirteen weeks ago he had a sore on his glans penis. He has had some sore-throat, and the skin shows pigmented remains of an old eruption. The patient is rather thin, but otherwise in good condition. Shortly after his admission the patient began to be delirious, his appetite, however, remained good, his bowels regular. With the delirium, which rapidly passed into stupor, he complained of severe pain in the occiput. Two days afterward involuntary emissions took place. His mental condition grew worse, the few days' delirium giving way to stupor, followed by coma, ending in death nearly four weeks after admission. He seemed to improve with calomel and iodide of potassium. The separate masses continued to grow. The skin over the testicles in two areas became the seat of extensive ulceration, and a new growth appeared on the cheek similar to the one on the forehead. During all this time the patient had no fever, and no change in the lungs could be found. Unfortunately the eye-ground was not examined, and no positive clew given to the nature of the cerebral complication. *January* 26, 1888.

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3. *Recurrent sarcoma.*

Exhibited by Dr. J. H. C. Simes.

Anna E., German, æt. 39 years, was admitted to the surgical ward of the Episcopal Hospital on October 18, 1887. The following history was obtained: Twenty-one months previous she first noticed a "pimple" at the lower part of the thigh over the outer part of the knee. This gave her no pain, and no further notice was taken of it at that time. The "lump," however, gradually increased in size, although it continued painless, and at the end of fourteen months from its first discovery it was about as large as a hen's egg. She now first sought the advice of a physician, who recommended removal of the growth. An operation was submitted to and the tumor removed. The wound soon healed and remained well for some time, when a rapid new formation of the growth occurred with ulceration and hemorrhages. An examination at the time of her admission into the hospital showed a large ulcer-

ating tumor, measuring six inches in length and four inches in width, seated at the lower and outer side of the thigh, extending over the knee-joint. The growth was painful and bleeding upon the slightest touch. While there was a perceptible movement of the tumor when siezed, it gave the impression of being attached to the structures of the joint, but not to the bone. There was no enlargement of the inguinal glands. The leg was flexed and could not be extended; any attempt at extension gave great pain. The surface of the ulcer was covered with a very offensive and purulent discharge. The general condition of the patient was not good; she had suffered much pain, had been sleeping poorly, and her appetite had failed. Three days previous to coming to the hospital there had been quite a large hemorrhage from the tumor. Amputation was advised, and on October 20th I performed the operation at the lower third of the femur. The result has, until the present time, been good; the patient has recovered her health, and the wound is nearly healed.

An examination of the limb after its removal shows the growth to involve the tissues about the joint, including the capsular ligament. A section of the tumor, made for microscopic examination, presents the histological structure of a large-celled sarcoma. *January* 26, 1888.

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4. *Degenerated sebaceous cyst.*

Exhibited by Dr. H. R. Wharton.

This cyst was removed from the forehead of a woman, æt. 30 years, a patient in the University Hospital. The patient had noticed a small growth on the forehead near the root of the nose for some years, and it was not until it had been scratched by her children's hands that it discharged and bled and began to increase rapidly in size. It has been about three years in attaining its present dimensions.

The tumor seems quite solid and presents several openings from which blood and purulent matter were discharged.

The growth was probably a sebaceous cyst which was ruptured, and from constant irritation has been converted almost into a solid tumor, and in appearance closely resembled a malignant growth.

The history of the case and the specimen show the changes which may take place in a sebaceous cyst when it is ruptured or irritated, the

normal contents being replaced by a solid growth from the lining membrane of the cyst.

Calcareous degeneration may also occur, or the ruptured cyst may break down into a very intractable form of ulcer.

February 23, 1888.

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5. *Sarcoma of thigh.*

Exhibited by Dr. H. R. Wharton.

The tumor was removed from the right thigh of a man, æt. 65 years, with the following history: The patient noticed a tumor situated on the upper part of the right thigh over the trochanter in April, 1887, which did not cause him much trouble until October, 1887, when it began to increase rapidly in size and its growth was accompanied with pain. The tumor was removed February 11, 1888, by Professor Ashhurst, and was found to be adherent to the fascia lata, part of which was removed with the growth. The tumor was probably a sarcoma, and if its point of origin was from the fibrous tissues of the fascia lata or connective tissue, it is probably of the spindle-celled variety, in which case the prognosis, as regards recurrence, is more favorable than in the case of round-celled growths.

This growth, I think, is of the class which has been described by Paget as recurrent fibroid. It may also be noted that true cysts are more often found in connection with spindle-celled growths than in connection with round-celled sarcomata; in this specimen the presence of cysts can be clearly demonstrated. *February* 23, 1888.

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6. *Multiple Sarcoma.*

Exhibited by Drs. de Schweinitz and Jameson.

P. S. was admitted to the Insane Department of the Philadelphia Hospital February 10, 1873; a male, white, a native of Ireland, and a shoemaker. He was a chronic mania case with considerable mental deterioration. He had an iron constitution, and was an indefatigable worker. Six months before his death he lost interest in his work, and complained of pain in the small of his back. The pain grew rapidly

worse and he lost flesh and strength, but his appetite remained moderately good.

Physical examination, February 21, 1888: Height, 5 feet 5½ inches; weight, 114½ pounds. No scars or ulcers appear on any part of the body. Muscles are small, but rather firm. Inguinal glands are somewhat enlarged, but the post-cervical glands are apparently not enlarged. Crown of the head is bald; the remaining portion of the scalp, the bearded regions of the face, and the chest are covered with gray hair. The head is regular in contour, and large. Ears and nose are apparently normal. There is no facial paralysis. Breath is very offensive. Teeth of upper jaw are in very poor condition; many are out and others are represented by stumps. The teeth of the lower jaw are fairly well preserved. Tongue protrudes in median line, is large, flabby, covered with a gray fur, and is slightly tremulous.

Chest is somewhat barrelled. Expansion is medium and equal on both sides. Vocal fremitus and resonance are good. Percussion gives vesiculo-tympanitic note over whole chest. Vesicular murmur is clear, and expiration is prolonged and low pitched. Area of cardiac dulness begins at lower border of fourth rib, extends down left border of sternum and across to apex-beat, which is not palpable or visible, but which is located by sound a half inch above the normal situation. Heart-sounds are clear at apex and free from murmur. Same obtains at aortic cartilage. There is a distinct harsh systolic murmur heard at the xyphoid cartilage. It is heard over an area of an inch and a half in diameter, and is not transmitted in any direction. There is a distinctly visible pulsation along the left chondral margin.

Area of hepatic dulness begins at the seventh rib, and in the nipple line extends about half an inch below the costal margin. Midway between the nipple and sternum there is dulness continuous with the liver dulness extending to the level of the umbilicus. In the median line dulness extends almost to the umbilicus. On the left side the dulness extends in a curved direction to the costal margin. On palpation, in the right hypochondrium and the epigastrium, there is a rounded tumor apparently continuous with and having the shape of the liver. On deep palpation there is a notch felt a half an inch to the left of the umbilicus. Over the entire area of dulness a forcible impulse is transmitted to the hand. The surface of the tumor is covered with numerous small nodules about the size of peas. These impinge distinctly on the hand during deep inspiration. The tumor is tender, deep palpation not infrequently causing the patient to cry out. On auscultation there is a powerful impulse transmitted to the ear, together with a loud systolic

bruit. Posteriorly there was some tenderness over lumbar vertebræ and great tenderness over the lumbar muscles.

March 30. Patient has grown rapidly emaciated, and has not been able to sit up since before previous examination. His appetite has been generally good, and he has had, on an average, one putty-colored stool per day. The area of hepatic dulness now extends about two inches below the level of the umbilicus. The edge can be readily felt, and some of the nodules appears as large as a quarter of a dollar. He constantly complains of pain about the tumor, and very much more severe pain in the lumbar muscles. When he is asked to move, he tries, but often halloos with the pain. Motion or pressure appears to produce a lancinating pain. On the night of March 18, 1888, patient began to spit blood, and on the following day both spat blood and passed it by the bowel. The largest quantity passed at one time was on that evening. It was easily half a pint.

22*d*. Patient is now very feeble, and appetite is absent. The hemorrhage has ceased. There is now a tumor on the right side of the scalp, and a slight contused appearance at its upper surface.

23*d*. Patient is in no way improved. A smaller tumor has appeared on the opposite side of the head.

30*th*. No improvement noted. Tumors on head are increasing in size, are soft, but not fluctuating. Abdomen is distended and tenderness is general.

Autopsy, twenty-two hours after death.—The body was that of an extremely emaciated old man. Post-mortem rigidity was well-marked. No scars were apparent. The abdomen was distended and tympanitic, and the walls were tense. Below the right costal margin there was a slight rounded elevation. On the right side of the scalp, in front of the line of the ear and two inches above the ear, there is a lobulated tumor about three inches long and two inches wide. On the opposite side of the head, on the same level and behind the line of the ear, there is a smooth tumor an inch and a half in diameter. When the scalp was removed the tumors were found to be subperiosteal. Adhesions of the membranes were so extensive that the brain had to be removed in the calvarium. The adhesions between the dura and the pia were readily broken up by the fingers, and the dura was left *in situ*. On the inner side of the calvarium in a position corresponding to the external tumors were smaller lower tumors, which, being pressed, would produce a movement of the outer tumor. These tumors were subdural, grayish-yellow in color, soft and friable. The right half of the occipital bone contains a number of similar growths about the size of a dime.

Brain: Weight, 52 ounces. In positions corresponding to the internal growths, shallow depressions are visible. Over the vault the pia is thickened and opaque; the bloodvessels dilated, tortuous, and dark. The vessels of the base are somewhat atheromatous, and the pia is opaque. The organ is firm, the lateral ventricles are dilated, and a number of small cysts are seen on the choroid plexus. Nothing else abnormal is detected.

Thorax: The under surface of the sternum contains numerous small growths similar to those of the skull. On the left sixth rib near each end there is an oval tumor, and at these positions the bone is wholly destroyed. A pedunculated tumor the size of a large marble rests on the diaphragm near the right border of the pericardium. There is another tumor resting on the right side of the spinal column immediately above the attachment of the diaphragm. This mass was similar to a segment of a sphere, of an inch and a half in diameter, cut through the third of its diameter.

A few old adhesions bound together the layers of the pleura at the right apex. Both lungs were enlarged and did not collapse. Under the pleura of both lungs many small hard masses could be felt, and on section they were found to be about the size of a pea, and resembled the tumor-structure in other parts of the body.

The heart was of normal size and showed a flabby muscle. The valves were all normal except the aortic, whose segments had a calcareous deposit along the attachment. The atheroma of the arch of the aorta was slight.

Abdomen: When the abdominal wall was cut through the intestines were bound together by recent lymph, and the cavity contained five pints of yellow fluid in which floated free lymph, and on being allowed to stand a thick yellow deposit formed. The peritoneal inflammation was general and probably most marked in the true pelvis. The liver extended below the umbilicus, and all over its anterior surface were many nodules, varying in size from an inch and a half in diameter to less than a half inch. These nodules had a saucer-shaped surface, the border of which was pink, due to the bloodvessels, and the rest cream-colored. The under surface had many similar but larger nodules. The gall-bladder was small, the wall was thick, dense, and grayish-yellow. There were no abnormal adhesions of the liver, and the capsule was not thickened but had some recent lymph all over it. The organ weighed six pounds three ounces.

Immediately under the liver there was a mass resting on the spinal column and extending from the diaphragm downward about six inches.

This mass consisted of the pancreas, enlarged lymphatic glands, and the stomach. The stomach was not enlarged, but a short distance from the pylorus there was a constriction. At this constriction on the greater curvature was an opening, but whether it was done by handling it is not certain. This ulcer was about an inch in diameter. The tail of the pancreas was not involved, and the head did not appear to be. The spleen was of about the normal size and character. The intestines were not involved.

The right iliac fossa was filled by a mass which, when removed, was found to be about three inches in diameter. It extended entirely through the bone and presented a small tumor on the external surface. The bone was wholly destroyed and but a band remained on the upper and anterior surface. The mass was soft, friable, and in the centre contained some pus.

The points of interest in this case were:

1. The excruciating pain in the back.
2. The absence of the gastric symptoms due to the primary lesion.
3. The cause of the peritonitis.
4. The rapid progress of the disease and destruction of bone tissue.

April 14, 1888.

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7. *Epithelioma of hand.*

Exhibited by Dr. H. R. Wharton.

The patient from whom this growth was removed, a woman, æt. 76 years, noticed a little more than a year ago a small ulcer upon the little finger, which gradually extended, destroying the finger and involving the hand to the extent seen. The growth was removed by amputation at the lower third of the forearm and the patient made a good recovery.

The chief point of clinical interest in this case was the age of the patient subjected to amputation for the removal of the growth. In the speaker's experience the proportion of recoveries in amputations in extreme old age is very encouraging. *September* 27, 1888.

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8. *A case of mediastinal sarcoma, with numerous metastases.*

Exhibited by Dr. T. S. Westcott.

By the courtesy of my chief, Dr. Meigs, I am permitted to exhibit the specimens of a very interesting case of generalized round-celled sarcoma.

Last year I presented to the Society the specimens of a case of the same disease which, while the primary growth was much larger, did not show the remarkable secondary results so beautifully exhibited by the present specimens.

J. C., æt. 21 years, born in Italy, came to the Pennsylvania Hospital October 1st, about noon, complaining principally of dyspnœa. Through an interpreter the following meagre history was obtained: Is a laborer and has been in this country eighteen months. He has not worked for forty days on account of headache and shortness of breath. Since June last has had chill and fever every other day. Dyspnœa did not become distressing until ten days ago. Urine is scanty and passed infrequently; bowels costive. Deglutition causes a burning sensation behind the sternum and brings on cough. Has had double vision for ten days.

Temperature on admission, 99°; respiration, 36; pulse, 92.

Physical condition.—Is a strong, well-developed man, of average height. The neck is extraordinarily broad and thick, and the head slightly retracted. The veins of the neck are enlarged and turgid, and the superficial veins of the chest are very prominent. Orthopnœa is constant and all of the auxiliary respiratory muscles act strongly. The face and neck are markedly congested and the forearms and hands are cyanosed. The lower extremities are not at all affected. The eyes are prominent and staring, and there is external strabismus of right eye. The tongue is protruded straight, is swollen, and yellow-coated. Some slight difference in the radial pulses of the two sides is noticed—the right being weaker and somewhat retarded. [This difference became very much more distinct during a paroxysm of suffocation, of which the patient had several before death.]

Physical examination of the chest showed the heart-sounds clear, sharp, and free from murmur.

Lungs: Anteriorly there seemed to be dulness behind the upper half of sternum and to the right over the area of the innominate artery, but this was not very clearly made out, and was indeed less distinct upon a later examination. Respiratory sounds seemed fairly good at apices and over upper lobes, but careful examination was prevented by the harsh, roaring dry sounds and the extreme distress of the patient. Posteriorly there was marked general impairment of resonance, which at bases amounted to dulness. This was more marked on the right side; and here tactile fremitus and vocal resonance were almost abolished and the breath-sounds very distant. All over the upper pulmonary area there are harsh, roaring breath-sounds, and whistling, dry râles, very loud at expiration.

Liver dulness extends about an inch below the costal border.

Splenic dulness very markedly increased.

The physical signs all pointed to intra-thoracic pressure, exerted especially upon the trachea, the superior cava, and the great vessels of the arch, especially upon the right side. Death occurred from suffocation about twenty-four hours after admission.

The *post-mortem* was held three hours after death. Upon removing the sternum and cartilages a large flesh-like tumor presented itself, occupying the upper half of the mediastinum and attached above at the root of the neck. This was of wedge-shape, tapering to a thin edge upon the anterior surface of the pericardium, and measuring about five inches in vertical direction by three inches horizontally, and reaching backward to the vertebral column. The trachea was compressed laterally. The arch of the aorta and its branches were completely enveloped in the growth. The innominate artery divided within the mass and its two branches were markedly narrowed, the carotid a short distance before its emergence showing a partial destruction of the wall. Upon vertical section the centre of the growth showed a channel crossing it transversely, which proved to be the innominate vein very greatly narrowed, as were its two formative branches at their entrance on the left side.

The *heart* was natural in size and its valves not diseased, but along the course of the coronary arteries, upon the external surface and at the apex, were deposits of the new growth. There were also masses upon the auricles and upon the roots of the great vessels; and at one spot, near the base, the growth extended to the parietal surface of the pericardium. The right lung was greatly compressed by an extensive blood-stained pleural effusion, and there was also a small amount of liquid in the left sac. Upon the diaphragmatic surface of the right pleura was a sessile mass of the growth, and very numerous deposits were seen upon the surface of the lungs, infiltrating their substance.

The liver was enlarged, very congested, weighing nearly five pounds. The spleen was five times the natural size and very firm and hard. Neither of these organs showed neoplastic deposits.

The kidneys showed numerous pea-sized nodules upon the external surface and throughout the substance. The mesenteric glands were greatly enlarged.

The brain was examined and was to all appearances healthy, except the oculo-motor nerves, which were enlarged, the right especially presenting a pear-shaped swelling just before entering the sphenoidal fissure.

Through the kindness of Dr. Meigs I am enabled to present a very complete set of microscopic sections of the numerous tissues in which we found the growth:

No. 1. The primary growth, showing small round-celled sarcoma.

No. 2. Another portion of primary growth, giving a cross-section of the right carotid artery and showing partial destruction of its wall and sarcoma cells in the lumen.

No. 3. Kidney, showing various large and small areas of neoplasm in the cortex. In the medulla numerous columns of cells can be seen advancing between the straight tubules of the pyramids.

No. 4. Section of papillomatous growth on the diaphragmatic pleura, showing infiltration of the muscular fibres of diaphragm.

No. 5. Section of heart through coronary artery, showing infiltration of the muscular tissue and complete erosion of the wall of the coronary vein.

No. 6. Section of growth at the apex of heart. Some slight infiltration of the peripheral muscular substance is seen.

No. 7. Section of the right oculo motor nerve. This seems to be a sarcoma matrix through which run the fibres, each one standing out prominently alone and not taking stain.

No. 8. Section of growth in lung.

No. 9. Another section of kidney.

No. 10. Section of mesenteric chain.

Dr. Meigs said: The patient was under my charge in the hospital, and the case presents no unusual features, except the involvement of the oculo-motor nerves, a thing of great rarity and interest. Death took place so soon after the man came under our care that there was no opportunity to make an extended study from the clinical standpoint. At the time of death I supposed that the immediate cause was the direct pressure of the tumor in the mediastinum, but since seeing the sections of the oculo-motor nerves I am disposed to think there must have been involvement of the pneumogastrics or of the medulla oblongata, for it seems almost inconceivable that the increase of size of the mass in the mediastinum could have been rapid enough to cause such sudden death. The man came in one day without any oppression that seemed very urgent, and then after paroxysms of suffocation of exceeding violence died the next. Unfortunately, this was not thought of at the time the autopsy was made, and therefore neither the pneumogastric nerves nor the medulla oblongata were either examined or retained. The extended microscopic examination of various parts involved brought to light some points of interest. The growth in the mediastinum, which was

probably the original seat of the disease, seems to be made up of a mixture of round-cell and alveolar sarcoma, and the way in which at one point the disease had destroyed the carotid artery, which ran through the tumor upon one side, so that there was nothing of it left except some slight remains of the fibrous coat, while at other parts of the circuit the muscular coat and intima were left intact, is curious. The lung also presents a mixture of the round-cell and alveolar formation. It is of interest to ask the question whether the minute aggregations of round cells which are so abundant in the liver, and which are so commonly found in that organ in different diseases, are in this case to be looked upon as the commencement of sarcomatous deposit? The spleen does not seem to me to present any appearances which, in the absence of a knowledge of the extensive disease of other parts, could be recognized as sarcoma. The involvement of the mesenteric glands was most extensive and the microscopic appearances presented very typical. The mode of invasion by the disease of neighboring parts is beautifully shown by the sections of the diapragm, the muscular tissue being at different depths in all stages of granular and fatty degeneration. The heart sections show that, although there was quite a thick layer of neoplastic growth upon the surface, it was almost entirely confined to the fatty tissue upon the surface, and that invasion of the muscular substance, although plainly evident at some points, had but just begun. It is interesting, too, to note that the large artery contained in the section from the middle of the ventricle is hardly at all involved by the disease, no sarcoma cells having as yet penetrated as deeply as the muscular layer, while at the same time many of the neoplastic cells can be seen in all parts of the smaller arterioles around and in the veins. The absence of extensive involvement of the muscular structure tallies exactly with what might have been expected, for during life the heart action seemed strong and effective. The oculo-motor conditions are of peculiar interest, the right nerve being of fully twice the diameter of the left. The right presents few spots where more than two or three nerve tubes can be seen together unsurrounded by sarcoma cells, and these generally the smaller ones, for in most places each nerve is encircled by a string of the neoplastic cells; the left presents the disease in an equally typical manner, but much less far advanced.

October 25, 1888.

9. *A case of melano-sarcoma.*

Exhibited by Dr. J. C. Wilson.

The specimens exhibited consisted of portions of both lungs, the heart and pericardium, the stomach, portions of the liver and intestines, the right kidney, the spleen, a large sarcomatous growth from the pelvis, a number of mesenteric glands, one mediastinal gland, and the stump resulting from the amputation of the left eyeball, together with microscopical preparations.

They were removed from the body of C. C., male, æt. 25 years, a laborer by occupation, who died in the Philadelphia Hospital November 28, 1888. Family history negative; patient addicted to alcoholic excesses for six years; had gonorrhœa three years ago; three months later an inflammation of the left eye, followed by total loss of vision; globe removed October 1, 1887, at the Wills Eye Hospital, by Dr. Strawbridge, through whose courtesy I have received the following brief note from the hospital records:

"*Diagnosis:* Rupture of left eyeball; history of anterior staphyloma, probably following perforating ulcer of cornea eighteen months ago. Three days ago patient received a blow upon the eye from a fist, producing hemorrhage and extreme swelling of eyeball, which is found to be ruptured, and protruding through the lids."

Nine weeks before admission to the Philadelphia Hospital the patient was much exposed during a debauch; his skin suddenly became sallow; had swelling of the feet; slight cough; no expectoration; no dyspnœa; troublesome diarrhœa.

Upon admission patient was deeply jaundiced. The *left chest* flat upon percussion, except at apex posteriorly; intercostal spaces widened and bulging; upon auscultation absence of respiratory sounds; very feeble and distant vocal resonance.

Right side: Normal resonance upon percussion throughout, and puerile respiration; no râles. *Heart* displaced slightly to the right; cardiac dulness extends about two fingers' breadth to the right of sternal border at the level of the fifth costal cartilage. Loud, coarse, pericardial friction sounds, audible over entire præcordial region, and along the lower costal border on the right side as far as the axillary line, where it was by degrees lost. No endocardial murmur could be made out.

Liver greatly enlarged, reaching to the line of the anterior superior spinous process of the ilium, where its lower border could be distinctly

felt extending to the left and upward; the border and its surface were distinctly irregular, and coarse discrete nodules could be felt. At a later period in the case, owing to their increase in size and the atrophy of the abdominal walls, these nodules became plainly visible. In the course of the border of the liver, slightly below and to the right of the umbilicus, there is to be recognized, both upon inspection and palpation, the oblong, oval, distended gall-bladder with the corresponding notch in the edge of the liver. No pain in the hepatic region; slight general tenderness on deep pressure.

The desire for food continues; no vomiting; bowels moved three to six times daily; passages clay-colored, greasy, and offensive. Area of splenic dulness not made out. Urine dark-colored; specific gravity 1014; reaction acid; no albumin; no sugar; no indican. Bile pigment abundant.

In the course of ten days pericardial friction-sound disappeared completely. There was then recognized faint systolic bruit at the base of the heart. At one time in the course of the case the urine deposited for a few days abundant crystals of tyrosin and leucine spores. The distended gall-bladder rather suddenly disappeared from view with recurrence of evidence of admixture of bile with the alvine dejection and some diminution in the area of liver dulness. Coincidently with this there was a slight diminution in the intensity of the jaundice. The patient's temperature, which had ranged between 102° and 103½°, gradually sank to a degree or so above normal, his strength failed progressively, and death occurred on November 28th.

Autopsy.—Body of a medium-sized man. Moderately emaciated. Deeply jaundiced. Left eyeball out. No enlargement of superficial lymphatics. There are no pigmented bodies on skin. Muscular tissue bright-red. Two or three small new-growths in rectus abdominalis muscle on right side, near pubic bone.

Omentum: Dark, deeply pigmented, and crowded together beneath left lobe of the liver, and containing at various points small, nodular new-growths of some size at its border, and studded throughout with minute growths, of a similar kind, like black sand.

Thorax: No pleural adhesions on right side. On left side firm pleural adhesions at apex, and more recent ones at base. No pleural effusion on either side.

Lungs: Right weighs four and a half pounds. It is crepitant throughout, showing upon pleural surface a number of circular, deeply pigmented, button-shaped new-growths, varying in size from 2 or 3 mm. to 3 or 4 cm., the larger of these being oblong. Similar growths are

scattered throughout the substance of the lung in relation with the larger bronchial tubes. These growths are deeply pigmented, in some instances being black, and almost diffluent in consistence. The left lung: Upper lobe enlarged, tense, infiltrated throughout with diffuse new-growth, which is deeply pigmented, and of moderately firm consistence. Lower lobe compressed, airless. The bronchial tubes dilated, filled with purulent fluid. The main bronchus is filled with deeply pigmented soft masses of sarcomatous tissue. The peribronchial lymphatic glands enlarged in size from a pea to a cherry, deeply pigmented, and of pulpy consistence. The bronchial glands at the root of the lungs enlarged and pigmented. Several of the anterior mediastinal glands enlarged. A large, nodular, irregularly pigmented, grayish mass, the size of an apple, apparently springing from an anterior mediastinal gland, occupies the position of the apex of the heart.

Pericardium: Universally adherent.

Heart: Displaced to the right, its border occupying a position an inch and a half to the right of the sternal edge. All the cavities contain fluid blood. The wall of the left ventricle thickened. The right ventricle rather small. All the valves normal. No new growths in heart or pericardium.

Aorta: Normal.

Portal vein: Moderately dilated; no new growth in it.

Liver: Greatly enlarged, weight 9¾ pounds; studded throughout with thickly set, irregularly round, and oval white masses, varying in size from the head of a pin to an orange. The larger of these occupy a position on the upper surface of the organ, along the suspensory ligament. The intervening liver tissue is dark, firm, and shows increase of the connective tissue. The gall-bladder is enormously dilated; hepatic, cystic, and common ducts all dilated. The gall-bladder and ducts contain a dark, greenish-black tarry fluid; no calculi. The hepatic ducts throughout the structure of the liver are dilated, and contain at various points a purulent fluid. Many of the masses in the liver substance have undergone purulent softening; several of them show points of pigmentation, and a few are pigmented throughout. A large sarcomatous lymphatic gland, the size of a small apple, in the meso-colon opposite the pyloric end of the stomach, presses upon the common duct, almost, but not completely, occluding it. A number of sarcomatous lymphatic glands are in contact with the head of the pancreas, but its structure is not involved in the growths.

Spleen: Small; weighs 4 ounces; shows upon its convex surface a number of small globular deposits lying just below the capsule.

Stomach: Slightly congested; otherwise normal, except at a point in the lesser curvature, where there is a single, oval, deeply pigmented tumor developed beneath the mucous membrane, and pendulous.

Intestines: Almost empty, and no involvement of the glands in the mucous membrane. Along the mesenteric border many small new growths, varying in size from a pea to a marble. Along the large intestine the appendices epiploicæ are uniformly enlarged at the end, and infiltrated with sarcoma.

Kidneys: Enlarged, capsule adherent. The tissue is firm, and on the cortex and throughout the substance show a number of areas of pigmentation. These are in narrow lines, looking as if they ran along the bloodvessels. This is much more extensive in the right than in the left kidney. The tissue is coarse-looking and bile-stained. The pelvis and calices of left organ much dilated. Left ureter also enlarged; below it passes directly between the growth and the bladder, and is here much compressed, but not completely occluded. On lower end of left kidney is a large globular mass, the size of a hen's egg, springing from the capsule. Both suprarenal bodies are normal.

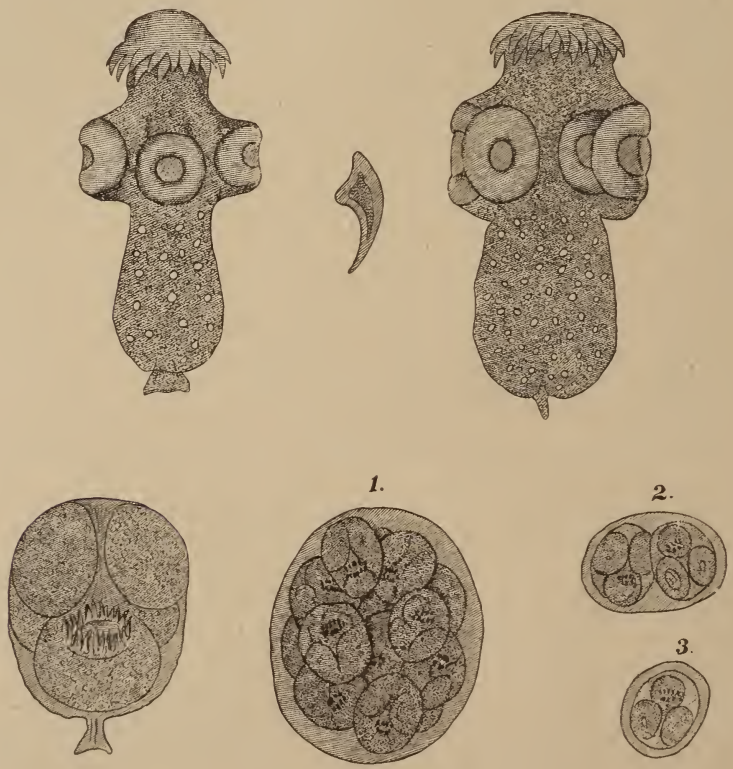
Lying between rectum and bladder, filling up the entire pelvis, is an enormous mass of sarcoma, the size of a cocoanut; with the rectum it is intimately associated. A large nodular mass projects into its cavity, covered only with the thin mucous membrane. The growth is also intimately united with the bladder, particularly at its base, from which, in fact, the growth appears to have originated. On section the mass is a soft dark sarcomatous tissue, into which in many places hemorrhage has occurred.

Brain: The dura is normal, bile-stained; pia natural-looking. On the ascending parietal, near the longitudinal fissure, there is a small pigmented patch the size of a ten-cent piece; on section it extended only about 2 or 3 mm. into the substance, and looked more like a deposit of pigment below the pia than an actual new growth. On careful section of the brain no other areas of disease found. At the base the left optic nerve was distinctly atrophic. The tracts looked normal. The contents of this organ removed; no sign of new growth but made up of a mass of fatty and fibrous tissue—the remnants of the ocular muscles—the optic nerve running through the centre of it. In one place only is there a little region of pigmentation. *December* 13, 1888.

10. *Echinococcus tumor in the abdominal walls.*

Exhibited by Dr. G. E. de Schweinitz.

Dr. Joseph Leidy has kindly given me the specimen I present this evening. The tumor was found by him some years ago while dissecting a lecture subject. None of the life-history of the individual was known, except that he had been an English sailor and was about twenty years of age. The growth was situated in the abdominal muscles of the right



iliac region, and was about the size of the fist. The mother cyst contained numerous secondary cysts. The largest brood sac contained twenty echinococci and this measured 0.45 or about one-half of a millimetre (see Figure 1). The smallest brood-sacs contained one and two echinococci. The sac containing one within it measured 0.135 in breadth, by

0.153 in length, and that containing two within it measured 0.225 in length, by 0.18 in breadth (see Figures 2 and 3). The average size of the folded echinococci was 0.135 by 0.108 mm. The circle of hooks was 0.045 in diameter, and the hooks 0.0222 long. The diagram represents three echinococci, a hook, and three brood-sacs. *December* 13, 1888.

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11. *Sarcoma of the anterior mediastinum.*

Exhibited by Dr. W. E. Hughes.

Annie M., æt. 12 years, had been a fairly healthy child when she was taken with what at the time was supposed to be mumps. This disease was epidemic in her neighborhood, she had been exposed to the contagion, and the first symptoms answered exactly to those of mumps—slight feverishness and general malaise, swelling first of one then of the other parotid, with inability to open the mouth wide. Instead, however, of the parotid swelling subsiding after a few days, it persisted, and it was this persistence of the swelling, rather than the general condition of the child, that led her parents to seek medical advice two weeks after the attack. Dr. J. F. Prendergast, the family physician, was called in and found the child slightly indisposed, appetite poor, bowels constipated, and having both her parotids uniformly and smoothly enlarged. While under his care she lost ground slowly and steadily, her temperature became slightly elevated, she developed a slight hacking cough, and finally began to expectorate a little bright-red blood. When I first saw her, five weeks from the beginning of her illness, she was slightly emaciated, anæmic, restless, and irritable. Her temperature was constantly above normal, ranging from 100° to 102°, with occasional profuse sweats. There was constant dyspnœa, it being impossible for her to lie flat in bed with ease. She had a constant, not very distressing cough, with an expectoration for the most part mucus, but occasionally mixed with blood, or more rarely pure blood. Within a few days there had been some hemorrhage from the gums, and at the time of the examination pressure on them caused them to bleed easily. The parotids had not changed since the initial enlargement. There was no enlargement of any lymphatic glands. The ankles were slightly œdematous and the knees swollen and tender. The only abnormality discoverable on physical examination was a soft, blowing, systolic murmur over the aortic area, the heart otherwise, as well as the lungs, being

normal. There was no albumin in the urine. A few days after this a profuse crop of purpuric spots developed over the lower extremities. She died two weeks later without any material change in the symptoms. There was nothing in the family history bearing upon the etiology of the case.

At the *autopsy* it was found that a sarcomatous tumor had developed from the lower part of the thymus gland, reaching the size of one-half that of a closed fist. In shape it was like an exaggerated thymus. It had evidently pressed upon the aorta, giving rise to the obstructive murmur noted during life. The whole gland was not degenerated, the upper part being healthy, the line of demarcation between the healthy and diseased structures being plainly marked out. There were no enlarged lymphatic glands. The lungs were atelectatic in spots, but nowhere could be found a point from which the hemorrhage could have proceeded. It was unfortunately not possible to make an examination of the parotid glands. To Dr. Prendergast, with whom I saw the case in consultation, I am indebted for the privilege of exhibiting these specimens to you. *January* 10, 1889.

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12. *Melanotic sarcoma of axillary region.*

Exhibited by Dr. H. R. Wharton.

The patient from whom this growth was removed was a woman, æt. 27 years, who was admitted to the University Hospital during the fall of 1888 with the following history: About a year before her admission she had a small growth removed from her right forearm just below the bend of the elbow. Six months after this time she noticed a lump growing upon her chest between the outer margin of the right breast and the axilla; this continued to increase in size, and at the time of her admission was about the size of the closed fist.

The growth was removed by Prof. Ashhurst, and upon gross examination appears to be a melanotic sarcoma. This is a form of sarcoma which, according to Ziegler, is one in which there is a deposit of pigment cells, which are deposited partly in the tumor cells, partly in the fibrous matrix and vessel walls. Clinically it is very malignant form of sarcoma. *January* 24, 1889.

13. *Lymphomata of the neck.*

Exhibited by Dr. H. R. Wharton.

The patient from whom these tumors were removed was a boy, æt. 4 years, who had the following history: About a year before the present growths were removed he was brought to the University Hospital, and Prof. Ashhurst removed several large tumors of the same character from the right side of the neck.

He was admitted again last fall, a number of similar growths having developed, and the growths which I show this evening were removed. It was found at this time that a number of growths had appeared upon the left side of the neck, and that the chain of glands extending below the clavicles of both sides was involved.

The growths are apparently hypertrophied lymphatic glands, but whether due to simple hyperplasia of gland tissue or to the presence of tubercle, I am unable to say. But as the general health of the child at the time of operation was excellent, and as after the wound was healed it was noticed that a rapid growth had occurred in all the glands, I am inclined to think the case was one of lymphadenoma or "Hodgkin's disease." *January* 24, 1889.

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14. *Two specimens of ganglion removed by excision.*

Exhibited by Dr. H. R. Wharton.

The first of these tumors, about the size of a walnut, was removed from the palmar surface of the index-finger of a man æt. 50 years; he stated that the tumor had appeared ten or twelve years ago and had increased very gradually in size. On examination it was found to be covered by skin and firmly fixed to the proximal end of the distal phalanx of the index-finger; upon pressure the sensation of fluctuation was communicated to the fingers. Prof. Ashhurst removed the growth, which appeared to have its origin in the sheath of the flexor tendon. The growth appears to be made up of numerous small cysts containing serum, and I think represents a form of tumor which originates upon the sheath of a tendon or is made up by a dilatation or pouching of the sheath of a tendon, and is known as a ganglion.

I also present another specimen of ganglion with the following history

A. R., æt. 35 years, received a fall from a distance, alighting upon his right foot, which received a severe twist, and on the following day he noticed a circumscribed swelling just below the external malleolus, which has persisted since the accident. When he presented himself at the University Hospital, about a year after the accident, examination revealed a tumor under the skin just below the external malleolus, which was movable, and was apparently connected with the perineal tendons.

The growth was removed by Prof. Ashhurst, and it was found to originate from the sheath of the perineal tendons, which were exposed in removing the tumor.

This tumor would be described as a componnd ganglion according to the classification of Erichsen, who describes this variety of ganglion as one in which the growth or tumor consists of a dilatation of the sheath of the tendon itself and often involves several tendons.

The simple ganglion he describes as that form in which the tumor originates upon the sheath of a tendon.

The contents of these tumors may be a thin serous fluid, or, on the other hand, there may be a thick gelatinous fluid, which was the character of the contents of this tumor. Occasionally, as a part of the contents of a ganglionic tumor, numerous melon-seed bodies may be found.

January 24, 1889.

IX. MISCELLANEOUS.

1. *Some further investigations on the malarial germ of Laveran.*

Abstract of an address read by Dr. Councilman.

This organism, first described by Laveran, has been met with in every case of malarial fever which the writer has observed. The organism is in high degree polymorphous, and ten tolerably distinct forms may be found in the blood. Some of these forms evidently represent different stages of development, and the connection between them is obvious. Others present such marked differences in form that no connection between them can be made out. Some of the forms are only found inside of the red corpuscles, and others are found free in the blood. The forms described are: 1. Non-pigmented, small, amœba-like bodies inside the red corpuscles. 2. Pigmented bodies larger than No. 1, also in red corpuscles. 3. Pigmented bodies about the size of red corpuscles. 4. Segmenting forms of the No. 3 body. 5. Small hyaline bodies which are formed by this segmentation. 6. A crescentic-shaped body with pigment in the centre, the horns of the crescent being often connected by a fine line. 7. Round or oval bodies which differ from No. 6 in shape only. 8. A pigmented body provided with numerous long, actively-moving flagellæ. 9. Actively-moving free flagellæ which are evidently derived from No. 8. 10. A pigmented body with an active undulatory movement of its periphery.

The first five forms are found only in intermittent fever, No. 4 only being seen in the blood during the chill period, and its presence is invariably connected with the chill. Nos. 6 and 7 are found in cases of malarial cachexia. The most interesting forms, and about whose parasitic nature there can be no doubt, are the bodies Nos. 8 and 9. These are generally absent in blood taken from the finger, but they may be found in any type of the disease. They are the only forms of the organism whose presence in the blood is not associated with a special type of the disease. They were found, however, in fifteen out of the twenty cases in which the blood of the spleen was examined. Of these twenty cases, twelve were cases of malarial cachexia and eight of intermittent fever. In the twelve they were found ten times, and in the eight

cases of intermittent fever five times. From this it seems probable that Laveran was right in considering the flagellate organism as the most important form of the parasite. The influence of quinine on the intra-corpuscular forms of the parasite is most marked. Doses of fifteen grains t. d. for two days in succession were found sufficient to cause them to disappear. The effects of the quinine were not so apparent in the other forms. The crescents were apparently not diminished in numbers in one individual after he had taken forty-five grains of quinine daily for seven days, and sixty grains daily for four days.

Dr. Osler said the thing which had struck him most forcibly in looking over this subject was the almost perfect unanimity which has prevailed among the different observers as to the appearance of these organisms. With the sole exception of the segmented form (No. 5), Laveran and the other early observers had described them all. His own observations, since the communication he had presented to the Society last year, had been somewhat limited. He had, however, made a series of observations upon the blood of fishes and birds since it had been stated that bodies resembling Nos. 1, 2, 3, had been found in the blood of carp and some water fowl. Professor Baird had offered him facilities for this work at Wood's Holl, and had kindly furnished him with forty-five carp. He had failed to detect any such organisms in the blood of these. In the blood of a goose sent him from Ontario he had found one or two pigmented bodies. It had been stated by Dr. McCallum, who had sent him the goose, that it had malaria; however, the bodies were not abundant, nor was the temperature of the goose elevated, nor, so far as he could make out, had it chills. Dr. C. had not figured one body, which is very peculiar, indeed—namely, a solid body in the centre of a clear space. It stains like a microörganism, varies in size, and although the body itself does not change in form, yet there are sometimes changes in the outline of the clear space surrounding it. They were somewhat abundant in one case only. One other point with regard to the clear bodies (No. 1): in five or six instances he had seen such bodies pass out from the corpuscle, remaining out and undergoing no further change of form. He was not altogether prepared to say what was the relationship of these bodies to the other bodies described. It has been claimed that similar changes can be obtained by special methods of treating the blood. The most important question is first to determine the relationship of the hyaline to the pigmented bodies and the possibility that the hyaline may not be directly associated with them. He was convinced that the pigmented and segmented bodies were merely different stages. He could fully confirm what Dr. C. said with reference to

the crescents. They are most peculiar and interesting bodies, occurring in the chronic cases and in those in which there had been no chills. Three weeks ago he had lectured on a case as one of mild typhoid fever; it had lasted eight or ten days with constant fever—up in the evening, down in the morning; slight enlargement of the spleen; no spots. His resident had examined the blood and found what he thought were crescents. The case got rapidly better, left the hospital, and returned in a few days with a distinct chill, with crescents in the blood, and a well-marked remittent fever. The mobile forms he had not seen nearly so frequently as Dr. C.; though he had not examined the blood from the spleen, they had been present in eight or ten cases. Nor had he seen free filaments nearly so often; when he wrote his paper he had not seen them at all. Since then he had watched the process of separation. It was out of the question to suppose that the crescents and mobile forms could come from degenerations in the stroma of the corpuscle, but that the hyaline forms resulted from such changes was not altogether improbable, and further investigations were necessary to determine this point. *October* 27, 1887.

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2. *Weigert's fibrin stain.*

Read by Dr. G. Dock.

In showing these specimens I have no intention of taking up the time of the Society by a discussion of the theory of staining in histology, nor is it necessary before this audience to make any detailed explanation of the processes involved in the specimens themselves. The stain is too new for any great additions to our knowledge to have grown out of it; but its possibilities for demonstration and investigation can be estimated by an inspection of the sections. Briefly stated, in the origination of this method, Weigert has carried out his principle of specific stains. Proceeding from a study of the method of Gram, which depends on the action of Lugol's solution as a differentiating and fixing agent, with alcohol as an extractor, Weigert used in place of alcohol another solvent of aniline dyes—aniline itself. By this he found a stain which affects only certain microörganisms in fibrin. It is the latter which I here show, the bacteria stain not having yet received any special study. Under the microscope I have fibrin from two sources—one a croupous membrane from the tonsil, the other a fibrinous exudate on the pleura

from a case of tubercular pleurisy. I was disappointed in getting specimens of croupous pneumonia and fresh thrombus, the latter of which is instructive, inasmuch as the blood-plates do not take the fibrin stain, proving what has been held by many, but not undisputed, that they are not coagulated material. As you will see, the fibrin appears in these specimens as bright-blue fibrils, meshes, and thick masses.

November 10, 1887.

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3. *Nævus of thymus gland.*

Exhibited by Dr. W. Osler.

J. C., æt. 63 years, was admitted to the surgical wards of the Philadelphia Hospital November 29, 1887. His history is as follows: Mother and father died of senility; one brother killed accidentally; one brother and sister living and healthy. He had always been a healthy man; drank beer moderately; denies specific disease. For nineteen years had had leg ulcers, the cause of which he attributed to an attack of erysipelas. On admission he had two small but unhealthy ulcers—one upon either tibia. These, notwithstanding their treatment, grew worse, until the man developed marked constitutional depression, with many of the symptoms of pyæmia. He died ten days after admission.

The *necropsy* revealed the following: The body is that of a good-sized, well-nourished man; ulcers on each leg; extensive nævus (hypertrophic) of lower lip; simple nævus of skin covering chin, right side of face, and right ear, extending also down the sternum, spreading and gradually fading into normal skin on either side of the chest. The tongue is much enlarged and is noted to be in an angiomatous (cavernous) condition. Immediately beneath it is a teat-like process an inch in length and an inch at the base, where it seems to spring from the tongue above the frænum and overlapping that structure. It is also a hypertrophic nævus. On removal of the sternum in the anterior mediastinum the fatty tissues are permeated by a dense plexus of veins, which extends a short distance into the neck, forming in fact a large nævoid tumor, covering over the arch of the aorta, the roots of the great vessels of neck, and the trachea up to the thyroid gland. On dissection this mass (which is now passed around) is seen to be composed of two distinct portions, and it represents a greatly enlarged thymus gland. The gland is enveloped in a capsule, under which some of the veins are

large and prominent. On section the whole gland looks like a dense angioma. Dr. Dock has kindly prepared some microscopical sections, which, you notice, present the appearances as ordinarily shown in cavernosus angiomata, with but few traces of the normal glandular structure.

On further examination of the body a plexus of veins is seen to closely surround the adventitia of the aorta. Thyroid not enlarged. Veins over it look full. In the heart the mitral valves presented a condition of acute endocarditis, the rest of the organs being apparently normal.

A careful search was made through Virchow's *Archives*, the Virchow *Hirsch Jarhsbericht*, *Transactions of the Pathological Society of London*, and many Pathologies, without finding a reference to such a condition of the thymus as here described. *December* 22, 1887.

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4. *Diabetes.*

Exhibited by Dr. A. A. Eshner.

Maggie H., æt. 24 years; born in Ireland; occupation, domestic. Her father and mother, as also four brothers and three sisters, are all living and healthy. One brother died at the age of eighteen months, the cause being unknown. One sister died in infancy.

When a child she had the ordinary diseases of childhood and small-pox. When fifteen years old she had measles, and during the attack caught a severe cold, and has been troubled with a slight cough ever afterward, which (she claimed) disappeared almost entirely every winter and as promptly returned in summer. Two years ago she caught another severe cold, and from this time had a constant cough, but other symptoms of pulmonary trouble were indistinct, if present at all.

One year ago she was a healthy young Irishwoman, and while carrying a barrel of ashes up the cellar stairs she slipped and fell, the barrel of ashes falling upon her. The barrel was removed from her in from two to three minutes. She went to bed, having been pretty badly bruised and shaken up, but only remained in bed for a few hours, and for the next three weeks was up and about, feeling perfectly well.

She soon noticed that she drank a large quantity of water and was troubled a great deal with thirst. She now rapidly lost weight and strength, although her appetite and digestion remained good. Soon followed a constant sweetish taste in the mouth and excessive itching of

the surface of the body, especially around the genitals. She grew gradually weaker, and the amount of urine passed daily rapidly increased, until June 15, 1887, when she was admitted to the surgical wards of the Philadelphia Hospital for scalded feet, made a good recovery, and was transferred to the medical wards August 14, 1887. Her cough had become much worse, although she expectorated but little and had no hemorrhages or night-sweats. Weight of body had fallen in less than a year from 152 to 90 pounds. On and about August 15th she was passing on an average twelve and one-half pints in twenty-four hours. Sp. gr. 1.042. At this time she was placed on Martinot's treatment, and by August 26th the amount of urine passed had fallen from twelve and one-half to six pints. She drank about two quarts of Martinot's prepared water in twenty-four hours, using this as her only beverage. Her general condition also improved markedly.

August 3d she passed exactly four pints of urine. Sp. gr. 1.040, and was much improved generally and perfectly comfortable. Quantitative analysis at this time showed $5\frac{2}{3}$ per cent. of sugar present. September 2d she was taken off Martinot's treatment for three days and was allowed to satisfy her thirst by drinking ordinary water. The amount of urine passed rapidly increased from four or five pints daily to fifteen to nineteen pints daily. Amount of sugar was also increased to 6 per cent., her general condition remaining the same. She was again placed on the carbonate of lithium and arseniate of soda water, and by August 30th the flow of urine had again fallen to five pints. About this time she was furnished with some sugar by one of the patients and the discharge soon shot up to nineteen pints, with an aggravation of all the other symptoms. Strict treatment again brought it to seven pints on November 1st. Sugar was again furnished her and the amount of urine increased to twenty pints, with her general condition much worse. At this time she complained of much pain in the left breast, and on examination a drop of pus was seen oozing from the breast just below the nipple. An incision was made, followed immediately by a free flow of thick, ropy pus.

Another point formed just outside the nipple line and in the same interspace (fifth). This was also opened and free drainage allowed. She was rapidly failing in weight and strength, and the amount of urine passed fluctuated between fifteen and twenty pints. This condition of affairs continued until December 8th, when, in a fit of coughing, some thick, ropy pus and a little blood were expectorated. This recurred on the three succeeding days, and she finally died December 11, 1887.

Autopsy.—Thorax: Right pleural cavity and right lung perfectly

normal, except being slightly emphysematous at the borders. Left lung was adherent throughout, it being impossible to remove it without taking the costal pleura. Pleura was in some places as much as one-half inch thick. In lower lobe was a large abscess, which had burrowed upward, perforating the fifth interspace in the nipple line. It also had communication with an old fibroid cavity, about the size of a walnut, at the apex. Pus was found in the larger bronchi, and on opening them up a passage was found directly into the abscess cavity; the other organs were entirely free from tubercular deposit, and there was nothing else worthy of note.

Kidneys were normal in size and appearance, and did not show amyloid reaction. *January* 12, 1888.

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5. *Abscess in the mediastinum rupturing into the lung.*

Exhibited by Dr. E. T. Bruen.

The patient from whom these specimens were removed was a middle-aged man. He suffered from violent dysenteric diarrhœa on admission to the hospital, but there were special chest symptoms as follows: To the right border of the sternum, from the third rib downward to the sixth, was a line of dulness which was about two to three inches broad; in other words, the shape of the outline was irregular, resembling a pine-apple. The larger area of dulness was downward toward the upper margin of the liver. There was no cough or expectoration, and the outline was considered to define a pleural collection of fluid. The question of hepatic abscess was also considered, but not entertained on account of the absence of physical signs indicating enlargement of the liver. A few days before the death of the patient a marked reduction in the area of dulness was noticed. The duration of the illness in the hospital was three weeks, and the patient had been sick for four weeks previous to admission.

Post-mortem.—The cardinal features of the autopsy were the detection of the most extensive ulceration in the large bowel.

Examination of the thorax disclosed a bunch of callus scar. Over callus the tissues were adherent to the sixth rib. The inner surface of the sixth rib was rough, a small piece of lead attached to it. Two small pieces of lead were found in the adjacent tissues. The heart was pushed downward and to the left. The pericardium was adherent, especially on the right side. There were adhesions between the lung and ribs on the

right side at the apex and base; between the third and seventh ribs dense bands of adhesions were found.

Between the inner surface of the right lung, right side of pericardium and diaphragm below, there was a huge abscess, which had penetrated the lung in the region of the middle lobe, and then entered the lower and upper lobe, almost from apex to base, not touching upper and back part of upper lobe, and lower and middle of lower lobe; these portions of lung were crepitant throughout. The left lung was nearly normal, somewhat congested (passive).

The abscess had apparently penetrated the liver 1½ inches, as a small sac, about large enough to contain several walnuts, was found on the surface of the dome of the large lobe.

It was thought that the abscess was embolic in character; and on account of the limited extent of the hepatic lesion, the view that the abscess had originated in the mediastinum, thence had invaded lung and liver, was considered most probable.

In retrospect of the clinical history it would seem that when the area of dulness of the mediastinal abscess diminished it might have at that time perforated the lung. It is also very strange that no pus was expectorated. The temperature chart was that of continued fever—never the irregular type indicative of suppuration. The patient himself dated his trouble to a period ten years before, when he had been shot in the chest; had saved some of the bullet, the rest being found inside the chest. However, the location of the lesions renders the probability slight of the wound having etiological relation. Moreover, the tissues adjacent to the spot where the bullet was found were the most nearly normal in the lung. *January* 26, 1888.

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6. *General miliary tuberculosis.*

Exhibited by Dr. E. T. Bruen.

Henry A., male, colored, æt. 49 years; died February 2, 1889.

Post-mortem notes.—On opening the cadaver general tubercular peritonitis was disclosed. The peritoneal folds and the investment of the intestines were thickly studded with miliary granules. The peritoneum was also markedly thickened. The mesenteric glands were enlarged and exhibited characteristic evidences of the tubercular process.

The diaphragmatic pleura was densely infiltrated with tuberculous

granules; the pericardium was adherent throughout. A large mass of tubercular glands was discovered in the anterior mediastinum over the pericardium. The pleuræ throughout on both sides of the chest were infiltrated with tubercle, but the parenchyma of the lungs was not involved.

The muscular substance of both right and left ventricle was hypertrophied and the cavities dilated. The right coronary valve of the aortic semilunar leaflets was the seat of atheromatous change, and some vegetations were recognized along the base of the valve in Valsalva's sinus.

Spleen: Several large areas of infarction were found in the spleen; the arteries leading to these areas of infarction were dissected and the points of occlusion recognized.

Kidneys: These organs were much enlarged, the surface smooth and white; capsule not adherent. On section several tubercular nodules were found in both kidneys, presenting a cheesy appearance.

Liver: The liver was found in a state of severe lobular cirrhosis.

Brain: Some pachymeningitis existed. The most interesting lesion was found in dissecting the posterior branch of the left middle cerebral artery, in which a small clot was found. There was softening of the cerebral substance throughout the entire motor tract on the left side; the softening was especially marked at the lower portion of the fissure of Rolando, near its termination at the Sylvian fissure.

Clinical notes.—The most interesting facts about this case are that the patient was admitted to my wards in the Philadelphia Hospital in October, 1887, suffering from dyspnœa, which, on examination, was ascribed to the effect of the cardiac valvular lesion (*i. e.*, aortic semilunar valves, as shown by the double murmur present), combined with renal disease, as shown by albumin with casts in the urine. There was no apparent pulmonary or peritoneal disease at that time.

In November he became hemiplegic with aphasia. During the months of November, December, and January he was under the care of my colleague, Dr. Wilson, and during these months the tubercular process must have developed. During these three months he never left his bed. Death occurred the second day after I resumed charge of the wards, in January of this year.

Another point of interest was that, although the lymphatic mediastinal glands were so much enlarged, no pain or reflex cough had ever been observed. *January* 26, 1888.

7. *Acardiac monster.*

Exhibited by Dr. G. Dock.

The mother was a healthy V-para. After the birth of a well-developed living female, and on attempting to remove the placenta, a solid mass was found presenting at the os, which felt like the buttocks of a five-months fœtus.

The sister is now eighteen years old and well-grown. There is no other case of monstrosity or deformity in the family, and no cause can be assigned for this. The specimen consisted of a larger extremity of the shape of the pelvis, measuring 9 cm. in transverse and 8 cm. in the antero-posterior diameter, and two stumps or legs 6 cm. long. The umbilical cord was inserted midway between the pubis and top of the body. Just above the insertion there was a button-like mass 2 cm. in diameter and ½ cm. high, covered with a cicatricial-looking skin. Beneath the pubis was a spheroidal body 2.5 cm. long and 2. cm. thick at the outer extremity, being thinner at its insertion. There was a raphe on the distal half of the inferior surface and a perpendicular meatus. This latter admitted a fine probe 2 cm. The legs ended in feet almost normal, outline and size corresponding to that of the pelvis. They ended in rudimentary great and (one each) small toes, showing nails. The skin for the most part appeared normal and was covered with lanugo hairs. A single vessel emptied from the cord, and, having divided frequently, soon became lost in the tissues. There was no bony pelvis nor any trace of abdominal or internal genital organs. The bones were slender and had no resemblance to normal leg bones. Owing to long preservation the specimen was not available for finer examination, but could be seen to be made up mostly of connective tissue, fat, and muscles. The specimen was one of Acephalus dipus, one of the lowest Acardiaci, and from its entire absence of abdominal and internal genital organs, one of the lowest of the genus so far recorded. There was as usual a single placenta, but this was not examined, so that the vascular supply must be left to conjecture. *January* 26, 1888.

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8. *Annual Address to the Pathological Society of Philadelphia.*

Read by Dr. F. P. Henry.

My first duty, omitted last year on account of my absence at the proper time, is to thank you, and I do so most heartily for the honor, the

greatest at your disposal, which you have conferred upon me. My next is to endeavor to fulfil the requirements of Article V., Section 3, of the By-laws, which are, that the President of the Society shall, "at the annual meeting for elections in October of each year, make an address to the Society, in which he shall describe (sum up) the work of the Society for the past year, and make such suggestions in regard to its labors and the mode of conducting them as may seem to him desirable."

In endeavoring to obey this rule I shall not enter into minute details with which your regular attendance at our meetings has made you familiar, but will merely call your attention to certain figures which demonstrate the flourishing condition of our Society. The average attendance at our meetings during the last year has been nearly 26; the largest 35; the least 14.

Eleven members have been elected, four have resigned, and five have been dropped for non-payment of dues. There are at present 175 members. Dr. G. Fedeli, of Rome, is the only new corresponding member. In estimating the average attendance the two semi-annual meetings have been omitted.

I would call your attention to the fact that since the organization of this Society, on October 14, 1857, there have been elected fifteen corresponding members, of whom nine are dead, making six the number now on the list. I would suggest that a certain limit be fixed to the number of corresponding members, whether this be ten, fifteen, twenty, or more, and that an endeavor be made to keep this list filled with the names of distinguished pathologists. I have reason to know that the honor of being a corresponding member of this Society is one that is highly prized.

Gentlemen, I know of no better instance of the frequent misapplication of terms in our language than that furnished by the title of the body over which I have still the honor to preside. We speak of the specimens laid on the table as pathological, and we apply the same adjective to the Society engaged in their study. Nothing could be more inappropriate, and yet so infallible in such matters is custom, that nothing better can be suggested. Turning from the consideration of the name to that which it misrepresents, I am sure that no scientific body could be found in a more healthy condition. Its circulation, as represented by the interchange of ideas, is regular, active, and vigorous. Its nervous system shows no signs of erethism or irritable weakness, and lastly, but not least, its alimentary system is supplied with abundant pabulum, which is so well digested and assimilated that the discharges of flatus are few

and far between. No better proofs, I am sure, could be offered of the fact that the organism is in a healthy condition.

Objections have been made to the study of pathological specimens on the ground that they represent, in concrete form, our sins of omission and commission with reference to the more practical matters of diagnosis, prognosis, and treatment. Nothing can be more absurd than such an argument. So long as man is mortal, that is, liable to be converted, so far as his body is concerned, into a mere subject, the study of the normal and morbid anatomy of his organs and tissues must form the basis of all scientific attempts to keep them normal and restore them when abnormal. In witness of the truth of these views I have only to ask you to recall to your memory the numerous specimens which plainly show the direction which must must be taken to arrive at a cure. This is seen in the deposit of fibrin in the walls of arterial aneurism; in the pericardial adhesions in cases of cardiac aneurism, and in the barrier of leucocytes raised against the invasion of micrococci in diptheria. Pleuritic effusions are, I believe, often conservative. Œdema of the lungs, which is so common an event in cases of Bright's disease, is favored by the obliteration of the pleural cavity, also commonly present in these cases; or, in other words, the pleural cavity serves as an outlet for effusions which, but for its presence, would infiltrate the lung parenchyma. I have been particularly struck with the dilatation of the ureter and pelvis of the kidney in cases of urethral stricture, thus forming a supplementary bladder. Such dilatation may be recovered from even when extreme, for it is the rule in pregnancy. I am sure that numerous other examples of conservatism in disease will occur to the minds of all present, and I believe I am quite safe in saying that no one could attend the meetings of this Society for one year without deriving much valuable information upon the diagnosis, prognosis, and treatment of disease.

Gentlemen, the law of the survival of the fittest will, naturally, always be upheld by the survivors, but, in so far as it applies to our Society, numbering, as it does, among its members, men of world-wide reputation, the exceptions are altogether too numerous to prove the rule. It is not the custom to read eulogistic memorials of deceased members of this body, and it is unnecessary, so long as we publish a volume of *Transactions* in which they may build up for themselves their most fitting monument, but on an occasion like this it is well to review the list of those who did good work while with us, and gave promise of doing much more. The names of Bertholet, Cathcart, Hare, Hodge, Hunter, Jenks, Little, Meigs, Parry, and Richardson are well known to everyone present, and many of you were well acquainted with the men who bore them. For

my part, I had the privilege of a more or less intimate acquaintance with every one of those I have mentioned, and I am confident that it would be hard to find better representatives of medicine and surgery in general, and, in particular, of that branch of it which we study in this room. For a thorough knowledge of pathological histology, Bertholet was distinguished among his contemporaries, and but for his premature death this knowledge would doubtless have been the basis of many valuable researches. I learned to know Cathcart while he was resident physician at the Episcopal Hospital, a post which he filled to the satisfaction of all concerned. His graduating thesis was awarded a prize by the University of Pennsylvania, and there is little doubt that by this time he would have achieved an enviable rank in our profession. The early volumes of our *Transactions* contain many valuable contributions from the pen of Horace Binney Hare, a man who, it is positively certain, was arrested in the beginning of a brilliant career. Lenox Hodge, a former President of the Society, was a distinguished surgeon at the time of his death. He was favorably known as an ovariotomist, although his work was done before the marvellous improvements in abdominal surgery had been introduced. Hunter was remarkable for that rare quality miscalled common sense, a most excellent thing in one to whom not only limbs but lives are confided. There probably has never been in this city a gynecologist who began his career better fitted by training and disposition to make it successful than William F. Jenks. Through the prize that has been so liberally endowed in his name he will do by proxy the work to which his life would have been devoted. In the department of ophthalmology Little was distinguished for painstaking accuracy. In his day and generation, just passed, John Forsyth Meigs was the peer of such men as the elder Flint and Alonzo Clark. It is not gratitude, although that is great, which makes me say that his remarkable keenness of observation and unwearying devotion to his patients made him a model physician. Parry's treatise on extra-uterine gestation, a pioneer of its class, is a fitting monument of his industry, while the work of Joseph G. Richardson, obscured during his life by the smoke of controversy, is now beginning to be clearly seen.

It was not my intention to refer to the labors of any living members of our Society, but a fact which seems to me anomalous impels me to do so. In a neighboring city, the seat of a great university, and the home of some of our best pathologists, there is no pathological society. I have only to say that one obstacle to its establishment—the want of material—would be removed if it possessed, as does Philadelphia, a coroner's

physician who, regardless of personal inconvenience, lays open the rich storehouse of his experience to his professional brethren.

In conclusion, I would call attention to a circumstance that has been deplored by every well-wisher of this Socicty, viz., the want of a regular publication of its Proceedings. The numerous medical journals of this country seem to be so plethoric of "copy" as to have no room for the valuable contributions we are able to supply. This fact appears to me quite as anomalous as the one to which I have just referred. I am glad to announce that this obstacle to our complete success is about to be removed. The *University Magazine* has not only offered to publish our Proceedings regularly, but has made good this offer by printing an instalment of them in its first number. If any further stimulus is needed to bring out our best work, it is supplied by the fact that it will be published by a journal which, judging from its first exemplar, bids fair to be one of the highest rank. *October* 11, 1888.

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9. *The pathology of actinomycosis, with record of cases and experiments; and observations upon human actinomycosis.*

By Dr. George A. Bodamer.

The present paper is a record of personal observations on actinomycosis which I commenced in 1882 in the pathological laboratory of the University of Pennsylvania under the guidance of Dr. H. F. Formad. Early in 1883 I demonstrated to the laboratory class the actinomyces, or the radiating fungus[1] peculiar to actinomycosis, and my inaugural thesis consisted of a record of some of my cases of that disease and of the description of the fungus discovered by myself. It was awarded the first prize in 1884 by the Medical Faculty of the University.

Hence, I am probably entitled to priority in demonstrating the actinomyces in the United States. It appears, though, that since I made my demonstration of the pathology of actinomycosis it has received but little attention until recently, although the disease itself has been known for a long time as one peculiar to cattle. It is known in this country

[1] Dr. Belfield, of Chicago, and Professor Law subsequently described the fungus in American cases.

under the name of "swelled head," and affects usually the jaws of the *graminivora*, particularly the ox tribe; less frequently it affects the tongue, the malady being then designated as "wooden tongue."

The disease has been recognized as a malignant one, and metastasis to internal organs is known to occur. It was also recognized to be contagious or infectious, as it appeared to be transmissible from one animal to the other by cohabitation.

As to the pathology of the disease different authorities in former times expressed most diverse opinions, and the affection was designated as osteosarcoma, cancer, scrofula, tuberculosis, osteoporosis, hyperostosis, fungoid stomatitis, sarcomatitis, etc. Some authors looked upon it as a kind of glanders or farcy of the ox tribe.

Only since Bollinger more closely studied the disease in 1877 and designated it actinomycosis, on account of a fungus, the actinomyces, found to inhabit this lesion, a uniform conception of the nature of the disease was formed by the veterinarians of all lands, and the definition and name as given by Bollinger to this disease was generally adopted.

Ponfick, in 1879, was the first who described actinomycosis in man. although the disease had been observed by Israel and described as chronic pyæmia one year previous. And as early as 1845 Langenbeck also described a case which was unquestionably actinomycosis.

In the animal the disease manifests itself as a primary disease of bones forming tumor masses, secondarily giving rise to fistulous openings toward the exterior, and finally metastasis to the internal organs occur, particularly the respiratory tract, where nodules are seen not distinguishable from miliary tubercles.

In man, however, the disease commences in the soft parts as fungoid suppurating granulations, which ulcerate and finally proceed to the bones, inducing in the latter a caries or necrosis with everlasting chronic suppuration, and sometimes leading to exostosis or hyperostosis of the affected bones. Most commonly the disease seems to commence as a pleurisy, which takes a chronic course, terminating in ulceration of the adjoining bones, viz., the vertebral column and the ribs. An interesting case of human actinomycosis which I observed lately will be recorded in the latter part of this paper.

I have had the opportunity of seeing nine cases of actinomycosis bovis in Philadelphia, and one case in Berlin during my stay with Virchow in the summer of 1883.

Unquestionably the disease must be classed with the group of granulation tumors, a group of new formations in which Virchow includes sarcoma, tubercle, syphilis, leprosy, lupus, glanders, etc.

The new formation of actinomycosis, which may be properly designated as actinomycoma, is histologically closely allied to sarcoma.

On account of a fungus discovered by Bollinger, as above stated, inhabiting the lesions of the disease I am describing, and to which Bollinger gave the name of actinomyces, because of the radiating structure of this fungus, the disease is considered a parasitic one.

I made also some histological observations and cultures of the fungus and inoculation experiments on animals.

I have found, however, the fungus said to be peculiar to this disease in substances outside of actinomycomatous lesions, and among other locations in ordinary caries of bone and teeth in man.

From these facts, and from the results of my experiments, I came to the conclusion that the actinomyces are only concommitant, and are diagnostic of but probably not the cause of the disease.

Case I.—Early in April, 1883, Mr. X., a butcher, whom I had requested to supply me with all kinds of diseased products in animals, as I was pursuing some comparative pathologico-anatomical studies, informed me that he had an ox affected by a disease which he called "swelled head."

Upon my request I was permitted to be present when the animal was slaughtered.

Mr. X. informed me that animals thus affected are found once in a while, but that it was customary in his establishment, as well as in other droveyards, not to refuse such animals, unless particularly noticed by the authorities.

The animal in question was twelve years old and showed signs of having worked hard; it was sold on account of the lesion in the jaw. The lesion referred to consisted in a swelling of the right sub-maxillary bone, the tumefaction nearly altogether protruding outward and downward. The left side of the lower jaw, as well as the upper jaw, the tongue and pharynx, and the mucous membrane of the mouth, with exception of one point to be referred to, were perfectly normal. The swelling was due to a tumor, painful on pressure, perfectly immovable, and consequently affecting the bone as well as the surrounding soft parts; in size, ten inches in length, four and one-half inches high, and three and one-half inches in width. In the skin covering the tumor, and in the portion corresponding to the lower border of the maxilla affected, there were five ulcerated spots, varying in size from one-fourth of an inch to one inch in diameter, which proved to be openings through which the probe could be introduced into the tumor to a depth of from one to four inches.

After the animal was killed, the skin removed, and the lower jaw detached from the rest of the head, closer examination showed the following: The swelling was a uniform oval tumor affecting the inferior maxillary bone on the right side in the region corresponding to the molar teeth, without affecting the latter, and extending, as stated above, chiefly downward and outward.

To the touch the tumor was quite hard in most places, while in others it easily yielded to pressure, showing that it was partly bony in character and partly made up of soft material. The teeth, as stated, were in perfectly good condition. Externally, near the gums, where the tumefaction began, there was a number of whitish patches or surfaces of indurated tissue, which were continuous with the tumor, but no ulceration was found to open in the cavity of the mouth.

The appended photograph represents the specimen in its appearance, or rather its shape, when fresh, reduced in size.

The tumor was then sawed transversely in several portions to study its structure. It was seen that a new growth resembling sarcoma involved the spongy tissue of the bone, invading and destroying most of the cancellated and part of the compact substance of the bone, the latter being destroyed completely in some places, and in other places leaving only a sheet as thin as paper. The new growth involved also the soft parts—the muscles, connective tissue, and even the skin—and giving rise to the sinus leading downward, as described above. Very instructive preparations were obtained in those sections where a tooth had been sawed through in its longitudinal axis, exhibiting its roots and socket. It was now seen that there were fistulous openings leading to the socket of the tooth not detectable from the outside, and which appeared as if healed-up ulcerated passages led directly to the diseased parts. The healing of these fistulæ had been accomplished probably on account of the drainage established to the exterior in the lower part of the tumor through the openings in the skin.

The cut surface of the tumor presented, further, the following peculiarities: On scraping there was a milky, ichorous juice, containing a few yellowish granules the size of small poppy seeds. To these granules I will refer later. Throughout the whole cut there could be felt islands and streaks of bony structure alternating with the tumor mass, which presented various appearances, in some portions being quite hard and fibrous; in others soft and friable, and in still others gelatinoid and even puriform.

Throughout the whole new formation, but more so in the softer parts, could be seen the yellow granules referred to. As a striking peculiarity

FIG. 1.

Represents the lower jaw detached and separated from the animal under consideration; it gives the aspect viewed from above. On the right side is seen the tumor mass in its actual shape and appearance and relations to the part. On the upper surface, along the outer border of the teeth, is seen a narrow, depressed surface, somewhat abraded and bordered by the remnants of the detached buccal mucous membrane.

FIG. 2.

Represents the same specimen viewed from below. At the periphery of the specimen are distinctly seen some nodes and openings.

I may note that these yellow granules appear to multiply post-mortem, when the specimen is preserved in its fresh state, for instance, in a cool place for a number of days. After scraping the yellow granules away, some new ones would again appear on the surface, projecting from the tissue, on the lapse of a day or two.

I omitted to state that the animal from which the specimen was taken was examined carefully in all its parts, and none of the organs or tissue of the rest of the body was found to be diseased, so that the disease was evidently a local one.

In all parts of the tumor, in the firm as well as in the soft, and in the purulent exudation of the liquefying parts, I observed the yellow granules referred to above. These granules proved to be made up mainly of a branching or radiating fungus which I subsequently identified with the actinomyces of Bollinger, and the lesion itself as actinomycosis.

Case II.—In June of 1883, while working in Professor Virchow's Pathological Institute at the Charité, in Berlin, I saw a specimen of bone disease similar to that described in Case I.

I learned that Dr. Israel, one of Virchow's assistants, was studying up this affection, and was pleased to hear that I was not mistaken in my observation, namely, that the specimen under consideration was one of actinomycosis bovis.

Dr. Israel kindly demonstrated to me his own preparation of the fungus obtained from the lesions, which, however, did not correspond exactly with the organisms detected by myself in the American specimen. But in my further studies I learned that Israel's fungi were identical with mine, and merely represented different stages of the same growth.

The specimen in question was from a cow of the Municipal Drove Yard (Berliner Stadt Vieh-hof),[1] the left lower jaw of which was affected by a large tumefaction in the region of the molar teeth, the new growth also affecting the latter. The tumor was about twelve inches long, and five inches in its greatest width, oval in shape, with nodular uneven surface.

[1] As far as I remember, animals affected by actinomycosis, which is quite a common disease in Germany, are not rejected by the German sanitary authorities. At this point I also would like to draw attention to a rule adopted by the Berlin Municipal Board of Health in relation to the detection of infective diseases in animals used for food. As a measure to prevent, for instance, the sale of pork infected by trichiniasis, the butchers are induced to surrender such animals to the Health Office under conditions very profitable to them. The owner of the affected carcass is paid the full value of the animal, and all extra expenses incurred from its surrender, thus earning at once the money which he otherwise would receive from the retail sale of the meat. Such measure is besides enforced under penalty of the law.

On sawing the tumor open the appearance presented was in many respects very similar to that described in the Philadelphia specimen, only that the teeth and the alveolar processes were much displaced by the invading new formation. Here, also, the spongy portion of the bone was preëminently invaded and, as well as some of the soft parts, substituted by large, nodular, sarcoma-like, or fibroid masses. In other portions there were abscesses and fistulæ filled with a puriform semi-liquid mass, and cavities filled with luxuriant bony exostosis, and with necrosed bone particles, in addition to an ichorous liquid. Throughout the new formation there were also seen numerous granules, such as I described in my first case, and made up of many bunches of the actinomyces fungus. Some of these granules were yellowish, others rather white or gray, and distinctly calcified, so that the pulpified new growth felt gritty, as if mixed with sand.

Some of the tumor formations extended beyond the boundary of the bony framework, and invaded extensively the surrounding soft parts.

The microscopical appearance of this actinomycoma, the description and culture of the fungus, as well as the details of other histological studies made in the Berlin laboratory in connection with this disease, I will give collectively later on.

Case III.—The specimen from this case was observed, as in Case I, during life. It was taken from a bullock, six years of age, in the Philadelphia slaughter-house of Mr. X.

The part affected in this case was also the lower jaw, no other organ in the body of the animal being affected, except the lungs, which were partly collapsed and partly enphysematous. I made a careful examination of all the viscera, the spinal column, and of some of the bones. It may, however, be noted that the animal was intensely emaciated. The actinomycosis was in this case left-sided.

The tumor, as in the other cases described, appeared to have sprung from the region of the alveolar processes, extending into the spongy portion of the bone, but only partially and in some places, so that a part of the marrow cavity was not affected.

In size the tumor was smaller than in Cases I and II. The tumefaction, including the bony framework, did not exceed three inches in width in its longest diameter and six inches in length. The new growth invaded in some places also the soft parts, inducing several fistulous openings.

A peculiar feature was presented by some of the teeth, especially the fourth molar tooth. Here was seen a fistulous opening leading from without into the socket of the tooth, and thence communicating with the

new formation within the spongy portion of the bone. The tooth appeared quite loose, and upon removal presented appearances such as shown in Fig. 3. There was at the root a number of nodules of the new formation, about the size of a pea, or smaller, which upon examination proved to consist of actinomycomatous tissue. Upon scraping away these new formations the roots were seen roughened, exhibiting some bony excrescences or small hyperostoses.

Fig. 3.

The floor and the lateral walls of the socket appeared, on the contrary, rather necrosed, and besides showed depressions corresponding to the excrescences upon the tooth. See Fig. 3. That these appearances were abnormal was proved on examination of the tooth and socket of the corresponding normal side, which was examined for comparison. The other appearances and characters of the new formation were precisely the same as in Case I, only they were much less extensive, exhibiting

also less softening and much less ichorous or liquefying changes. Actinomyces granules were found here and there scattered throughout the affected tissue, but these were not so abundant as in the specimens before described.

Case IV.—The specimen was from the right jaw of a cow and obtained from the Philadelphia Abattoir. I could not obtain information concerning the age of the animal from which it came, nor whether there were any other lesions in the body. The specimen was also somewhat mutilated in its soft parts and broken or cut in half with an axe; still it represented an evident and typical case of actinomycosis.

The tumor invasion in this specimen was very extensive, although apparently not invading the teeth, which were left intact in the specimen. (Some of the teeth were broken out and the alveolar processes damaged when I received the specimen.) Otherwise the specimen was in every respect similar to that which I obtained in Berlin, and with lesions perfectly identical with those of the latter. Actinomyces granules were found abundantly, many of them being calcified.[1]

I have seen and studied six more cases of actinomycosis bovis in Philadelphia, all of which were analogous to those above described, the actinomyces being found in all of them. To save space I will refrain from giving details.

The Morphology of the Lesions. *Microscopical studies of the specimens from the above cases.*—From the character of the specimens, their naked eye appearances and the fungus which I detected in clusters making up the mentioned yellow minute bodies, I was fully aware that I dealt with lesions from a disease designated as actinomycosis. All the cases, however, which came under my observation represented only the local lesion of this disease.

It is well known now that the disease also generalizes itself, the primary new growths spreading by continuity of structure and, by metastasis, affecting internal organs. This generalization appears, however, to be the exception rather than the rule from what I could learn in my own studies. The new growth which I described above and of which I am about to give the histological details, must unquestionably be classed

[1] Since writing this paper Mr. X. sent me a new and magnificent specimen of actinomycoma of ox (fifth case). The tumor in this case is of the size of a man's head and affects the left upper jaw and evidently also the orbit. Some of the teeth are necrosed and fistulous openings lead to the interior of the tumor. The tumor otherwise appears to be identical with the specimens described above. A difference in relation to the affection of the teeth may be noted between my cases and those of Dr. Belfield, of Chicago. Dr. Belfield does not describe any lesions of the teeth in the animals affected.

with the tumors, being closely allied to osteosarcoma, with which it was formerly identified. It might be properly called *actinomycoma*. All the cases, including that met in Berlin, are in all their chief features perfectly analogous.

The microscopical studies also reveal their full identity so that the description of one will suffice for all. From the naked-eye appearance, and from physical properties, it was seen that there were at least five distinct morphological constituents of which an actinomycomatous tumor was made up:

1. The tissues forming the boundary of the tumor.
2. The bony framework.
3. The new growth proper in its solid parts.
4. The liquefied products.
5. The yellow granules scattered throughout the whole of the above constituents.

1. *The tissues making up the boundary of the tumor.*—These were not distinct. The new growth gradually shading off into the bony parts or else the integuments formed the boundary in such places where the bone was completely absorbed. The surrounding soft parts were, as a rule, much atrophied, and, as the microscope showed, infiltrated by cells from the new growth. In some specimens parts of the tumor masses were observed to be encircled by dense masses of connective tissue. These connective-tissue bands were seen on section sometimes to penetrate into the new growth. Some parts of the tumor were occasionally seen to project outside as lobulated fungoid masses. As a rule, however, the surface of the tumor was smooth. Contrary to my expectations, I found the yellow granules composed of masses or tufts of fungi, even in the most peripheral portions of the tumor. The capsule showed one large and several small clusters of the fungus imbedded between the connective-tissue fibres.

2. *The bony framework*, being similar to myeloid sarcomata, it appears to me that while the new growth is formed within the bone structure and at the expense of the latter, the bone also grows by opposition. The bony framework forms round or irregular spaces of various sizes, and while these spaces enlarge the bone tissue seems also to grow, and even to send bony excrescences or exostoses into the cavities filled by the new growth; I thought it possible that these apparent exostoses might be remnants of unabsorbed new-formed bone tissue. Often spicules of bones are met with in the soft tumor masses. Upon making section of the bony excrescences, I found that they had the structure of young callus tissue. Large osteoclasts, such as are found in the repair of bone,

or callus formation, are seen to make up the bulk of these structures. The cells are imbedded in a granular matrix, such as is common in young callus tissue before complete ossification sets in. Here and there are seen islands of mature bone tissue.

From the structural appearances of these bony excrescences it is evident that they are not remnants of bone tissue, but rather new formations. This tends to show that the new formation of bony or callus tissue is a part of the tumor formation in actinomycosis.

3. *The new growth proper in its solid parts.*—I have here reference to the nodular formations usually lobulated in character which formed the softer portions of the tumor. Sometimes, indeed, they are nearly semi-liquid; it is these parts which by means of fatty degeneration gives rise to the liquid parts of the tumor.

The appearance of this substance is white, often glistening, sometimes of milky appearance, in other places it is quite congested and red, and even specks of ecchymosis are occasionally seen.

Under low magnifying power a section presents granular appearance through which bands of connective tissue pass in various directions dividing the tissue into many partitions. In some places the granular appearance predominates, particularly so in the centre of the nodes, while at the periphery the structure has nearly a fibroid appearance. Under higher magnifying power the appearance of the new growth suggests very much sarcoma, either in its round- or spindle-cell variety; in other portions suggesting alveolar sarcoma, and in still other dense masses of connective tissue are seen into which the younger cells gradually merge.

4. *The liquefying products*, or a creamy liquid derived from the necrotic changes of the solid parts, were quite abundant in nearly every specimen; the centre of the larger nodes and sometimes the whole of a large node had undergone this change.

Under the microscope a small particle of the semi-liquid material, well teased, often showed quite distinct alveolar connective tissue intermingled with leucocytes, actinomyces, and the elements of softening necrosis.

5. *The yellow granules* scattered throughout the whole of the above-described constituents of the actinomycoma are clusters of the fungi which inhabit this new growth.

The fungus forms a necessary part of the anatomy of the new growth, or at least is extremely common. All my cases contained the fungus. On the other hand, if the fungus had not been present the new growth would have to be called sarcoma rather than actinomycoma.

No definite distribution of the fungus can be made out, the yellow granules, which represent the actinomyces, and which are easily distinguishable by the naked eye, are, as a rule, seen everywhere throughout the specimen.

The fungus is found more numerous in the liquefying parts, and here more often in small fragments, so that if a drop of liquid is taken and examined microscopically a large amount may be seen. In most instances masses containing the fungus were calcified.

Inoculation Experiments.—That the disease is transmissible from one animal to another there is now no reason to doubt, as the fact has been experimentally demonstrated by Johne and Ponfick. The experiments of these and others had previously failed, probably because the inoculation material was too old and had undergone change.

Johne[1] subsequently employed quite fresh material, and was successful in three out of four experiments—the animals being two calves, a cow, and a foal. The latter remained unaffected. The calves were inoculated subcutaneously behind the lower jaw and elsewhere, and a small quantity of the same material which was derived from a tumor of a living cow was also introduced into the peritoneal cavity. In one case death took place forty days after inoculation. The calf having lost its appetite, became emaciated and debilitated, and then succumbed. At the seat of inoculation, as well as in the abdomen, actinomycosis was markedly developed. In the second case death took place 114 days after inoculation, and the results were found to be as marked as in the other instance. The third case was a pregnant cow, which gave only a small quantity of milk. This animal was inoculated through the milk duct of the teat. The inoculation was in a few days followed by inflammatory œdema, which soon became developed into phlegmonous mastitis. Without any treatment the inflammation subsided, but there remained a small, hard swelling which increased so much that in three months the quarter of the gland was double its normal size, and felt like a hard fibroma. No milk was secreted. The cow was killed 133 days after inoculation, and in the udder were discovered all the signs of actinomycosis; diffuse fibroma, with variously located multiple spongy fibro-sarcomata, the interspaces of which contained the characteristic nodules or "granulation tissue," inclosing the fungus.

Ponfick[2] did not succeed in inoculating dogs or rabbits. He endea-

[1] Johne: Actinomykosis Bericht über d. Veterinärwesen im Konigreich Sachsen, 1881.

[2] Ponfick: Die Actinomykose der Menschen, eine neue Infections-krankheit. Berlin, 1882.

vored to produce the disease in cattle by feeding them with infective material in the form of fresh nodules, but the results were negative. By subcutaneous inoculation and intra-venous injection, however, he was completely successful, and the lesions of the former were similar to those in Johne's cases. Injection of the material into the jugular vein produced, in the course of two or three months, typical new formations in the lungs. The details of these experiments are very interesting, but too voluminous to be quoted in full. But they by no means conclusively prove the identity of the artificial with idiopathic disease, save the presence of the actinomyces, which was found naturally because it had been introduced.

There is no record of instances which might tend to show that the disease may be accidentally transmitted.

Ponfick relates the case of a woman, 34 years of age, who was attacked by the disease after having been for several years employed as a servant, in which position she was frequently among sick cattle, and these were affected with what the veterinary surgeon who attended them called "wurm," the popular name in Germany for the malady under consideration. This is the only case of probable transmission recorded.

To test the specific properties of the actinomyces fungus or materials containing it, I made the following experiments, strict antiseptic precautions being taken while the animals were kept separated and under the best hygienic conditions:

Table of Experiments.

No.	Date of inoculation.	Animal.	Inoculation.	The effects produced, and termination.	Result of post-mortem and microscopic examination.	Remarks.
1	Nov. 28, 1883.	Rabbit.	Inoculation made in left sub-maxillary bone and on the spine in the lumbar region cutting down to the bone. Inoculation material taken from the soft part of an actinomycoma of ox from Case III.	Wound healed at once. The rabbit did not appear ill, but on the third day swelling appeared on the seats of inoculation which on the fifth day formed ulcers and discharged pus. Subsequently cheesy lumps appeared which persisted up to the death of the animal which took place forty-three days after inoculation.	Animal was emaciated, cheesy tumors of the size of pigeon's eggs at seat of inoculations, but not complicating bone; actinomyces found abundant in the cheesy mass, but not in the surrounding parts. Lungs full of miliary tubercles, also *liver;* lymphatic glands enlarged; rest of organs normal.	It must be noted that no real actinomyces lesion was produced, but only a cheesy node, which served as culture soil for the fungus introduced. Tuberculosis was the result in internal organs mentioned.
2	Nov. 28. 1883.	Rabbit.	Inoculation made in angle of jaw and in spinal column in lumbar region with material from Case III. of actinomycoma.	Wounds healed, and formed cheesy masses of the size of a walnut at seat of inoculation.	Rabbit died January 10, 1884. Autopsy showed the lesions to have remained local, being expressed by the cheesy mass. Actinomyces was found, but only peripheral in the wound, while the cheesy mass proper was free from them.	
3	Nov. 28, 1883.	Rabbit.	Inoculation in thigh and angle of jaw.	Wounds healed and formed cheesy masses of the size of a walnut at seat of inoculation.	Rabbit died January 15, 1884. Otherwise same as experiment No. 2.	
4	Nov. 28, 1883.	Rabbit.	Inoculation made in spine at the neck.	Wound healed without result except small cheesy mass.	Rabbit died February 1, 1884. Organs full of miliary tubercles, nothing else peculiar. No actinomyces found.	
5	Nov. 28, 1883.	Rabbit.	Inoculation made in the neck on the spine as in the last case.	Wound healed, cheesy mass remained.	Rabbit died February 15, 1884. All organs full of miliary tubercles. No actinomyces.	
6	Jan. 8, 1884.	Dog.	Inoculation made on spinal column cutting down to the bone and also scarifying the bone. The matter used was taken from soft parts making sure that it contained the fungus.	Wound did not heal, but after three days became phlegmonous and indolent. The skin and subcutaneous tissue very much indurated. The deep structure, *i. e.*, the muscle and bone, seemed to be very much affected; there is swelling of the parts and hardness. The contents of the swelling is drained through a fistulous opening which throws off continuously a puriform liquid.	The dog is otherwise well, and is kept for further observation. The pus drained from the fistulæ contains actinomyces.	

TABLE OF EXPERIMENTS—*continued.*

No.	Date of inoculation.	Animal.	Inoculation.	The effects produced, and termination.	Result of post-mortem and microscopic examination.	Remarks.
7	Jan. 8, 1884.	Large cat.	Inoculation made with same material as in Case V., in the thigh and angle of jaw.	Wounds healed and an indurated node formed subcutaneously, which at first enlarged, but now is gradually subsiding.	Observation is continued.	
8	Jan. 8, 1884.	Small cat.	Inoculation made with same material as in Case V, in the thigh.	Wound healed rapidly, and cat became emaciated. Cat died February 28, 1884.	Autopsy showed a miliary nodular eruption in all its organs. These nodules did not contain fungi of any kind, and proved to be genuine tuberculosis.	
9	Jan. 8, 1884.	Cat.	Inoculation with same material as in Case V., in angle of jaw.	Wound did not heal, and a fistular opening communicating with an abscess cavity below.	Pus from fistulæ contains actinomyces Observation continued.	
10	Nov. 30, 1883.	Dog.	Inoculation in jaw with material from Case III. of actinomycosis bovis.	No effects, etc.	Died December 20, 1884. No lesions found.	
11	Nov. 30, 1883.	Rabbit.	Same as last.	Matter from fistulæ for a few days showed the fungus when gradually all healed up.	Died January 12, 1884. No lesions found.	
12	Nov. 30, 1883.	Rabbit.	Inoculation with cultured fungi obtained from infusion of corn, made in spinal column of neck, scraping the bone.	Wounds healed on third day, but soon cheesy lumps developed, identical with those lesions met with in the inoculation of fungi derived from the actinomycotic lesions. Animal died January 20, 1884.	Autopsy showed no internal lesions; the bone at seat of inoculation was found necrosed. The swelling consisted of an organized mass made up of connective tissue and lymph cells in state of cheesy degeneration and containing the fungus.	The fungi obtained from the infusion stated were in all respects identical with the actinomyces found in the bovine disease.
13	Nov. 30, 1883.	Dog.	Same as above.	Wound healed.	Animal was killed March 1st, but no lesions found on autopsy.	

My experiments show that inoculation of animals with materials from actinomycosis was followed, so far, by a formation of marked lesions in six out of thirteen animals.

One of the positive experiments showed the identity in the effects of fungi resembling actinomyces but found in the matter outside of diseased products (corn). But the experiments, I confess, are not altogether satisfactory, not being repeated in sufficient number. One thing they

prove, however, that the lesions produced by inoculation do not differ from tuberculosis except in containing instead of the tubercle bacillus the actinomyces fungus or no fungi of any kind. The presence of the actinomyces is not surprising, as I have already expressed above, as that fungus was introduced.

Control experiments with other than actinomycosis material were not made because I had witnessed in the investigations of Dr. Formad very numerous experiments with simple irritants to terminate in precisely the same manner as my above stated experiments; and, furthermore, I have seen that the lesions of induced tuberculosis have precisely the same appearance and manifestations as those obtained in my experiments with material from actinomycosis.

MORPHOLOGY OF THE ACTINOMYCES.

This fungus was discovered by Professor Bollinger in 1877, in a tumor from the jaw of an ox, and Dr. Harz, Professor of Botany at the Munich Veterinary School, gave the above name on account of the radiating arrangement of its mycelia. There is, however, evidence that Langenbeck, in 1845, had seen and described a fungus similar to the actinomyces, which he found in a case of necroses of bone in man. The morphology of this fungus has been also well treated of in the writings of J. Israel, Ponfick, Johne, G. Fleming, O. Israel, and others. (See Bibliography.)

It is sufficient here to say that the actinomyces is a large mould fungus (hyphomycetis), closely allied to the ordinary ubiquitous green mould, *Penicillium glaucum*, but differing from it, and, in fact, from all other known moulds in the radiating arrangement of the mycelia, as seen in fully developed tufts.

The individual adult plant consists of a round or oblong tuft of club-shaped mycelia, arranged somewhat after the manner of a bunch of grapes, except that the mycelia or hyphens of the fungus are closely packed, and radiate from one central root-cell in a star-shaped manner.

In its undeveloped state, in which it often persists, the actinomyces presents a single club-shaped mycelia, or in the form of conical figures of two or three mycelia springing from one root-cell, and presenting, as it were, abortive individuals of the fungus.

The majority of these tufts are aggregated in mulberry-like masses of from 0.5 to 1 millimetre in diameter, and appear to the unaided eye as

very minute dull-white or yellowish granules. Very frequently the tufts are somewhat calcified, and then it is difficult to make out their composition. By slight pressure made upon it the fungus tufts are considerably altered in appearance, and mostly assume the shape of a spheroidal segment, wherein some of the organisms can be distinctly traced throughout.

The actinomyces is the only fungus belonging to the class of moulds that have been found in the interior of animal tissues, such as the bones, and occurring also in the metastatic deposits in the lungs and elsewhere.

It might be said in general that there is no difficulty in detecting the fungus, on account of its large size and peculiar shape. Staining with aniline dyes is, however, advantageous, as the actinomyces retain the aniline dyes about as well as the tubercle bacilli. I found it most advantages to use the bacillus dye for specimens to be demonstrated.

Detection of the Fungus in Matter other than Actinomycosis.—1. Actinomyces in necrosis of teeth in man. I discovered, accidentally, the actinomyces in the pus from an abscess of the alveolar process of the superior maxilla. A specimen of pus was examined on many occasions from a necrosed tooth of this locality. The man upon whom the observation was made had, on account of annoyance from a gum boil, a hole drilled through the gums, superior maxilla, into the root of the tooth, which was necrosed, and this was discharging pus at intervals for at least two or three years. A preparation of this pus showed, on repeated occasions, a very distinct radiating fungus, which was also seen by Dr. Formad, and which we fully identified with the actinomyces.

2. Actinomyces was also observed by me twice in pus from necrosis of left submaxillary bone in a woman who was suffering from that affection. The fungus did not appear fully developed in either of these two cases, but when put into culture well-developed typical tufts were seen.

3. I detected the actinomyces in the scrapings from the tongue, tonsils, and mucous membranes of mouth in a number of cattle. Microscopical examinations were made of fifteen head of cattle, including oxen and cows. I made daily examinations, selecting a time of from twelve to twenty-four hours after they had been fed, expecting by this time to allow the mouth to act as a culture soil for the fungi that might be retained between the teeth or adhere to the mucous membrane of the mouth and tonsils. In five out of the above-mentioned fifteen animals very striking examples of the actinomyces were discovered.

The fungi were found mostly adherent to particles of straw, chaff, or corn adhering to the mucous membrane or sticking between the teeth.

4. I found actinomyces in particles of animal food.

(*a*) Bran, or the outer coats of grain, revealed the fungus in considerable quantity. An infusion of bran was made and examined microscopically a few hours after standing, when several small fungoid bodies represented in Fig. 5 were seen. At first not much stress was laid upon this, but subsequent examinations fully revealed their identity with the actinomyces. Repeated examinations of these infusions were made daily, and every now and then fragments of the fungus were detected, and where care was practised in making and cultivating these infusions fully developed actinomyces were occasionally observed.

(*b*) Examinations of the siftings of grain. Small fragments of grain were taken from the bottom of a large chest in which grain was kept in a barn (corn, mostly) and infusions were made of this material. In these infusions I found several times distinct fragments of the actinomyces as represented in Fig. 5. Examinations were kept up daily for two or three weeks, occasionally with positive, more often, however, with negative, results.

(*c*) Examination of chaff or husks of grain also revealed the fungus. Of these husks infusions were made as before. I observed on several occasions large radiating masses of the actinomyces, and fragments of crushed tufts in all forms and shapes were frequently found. Most satisfactory results were obtained by agitating the dry chaff in a glass bottle and adding to a small portion of this a drop of distilled water.

(*d*) In smut from corn the fungus was found, but in some cases I could not positively identify the actinomyces. I made cultures of this, but the results were doubtful.

(*e*) Fodder and pith of cornstalk were examined, together with scrapings from the outside of the stalk. Small pieces of these were shaken up in distilled water and immediately examined. I saw fungoid bodies in this infusion that I could not tell from actinomyces, and believe they were the fungus in question.

Most striking specimens were seen whenever the fodder was moistened and allowed in a moist chamber to partly undergo decomposition.

The common occurrence of the actinomyces in the food of animals, as proven by my examinations, suggests that this fungus is only a concomitant in actinomycosis, and further suggests that the fungus should be looked for in the lesions of other animal ailments.

The Isolation and Cultivation of the Actinomyces.—Cultures were made in strict pursuance with the usual well-known methods in bacteriology in a well-equipped laboratory. All known culture soils, but principally potatoes, pepton-gelatin, agar-agar, and blood-serum, were

repeatedly tried, but only the latter, coagulated in the test-tubes, was found available and useful for raising good crops. This, I believe, was also the experience of O. Israel, in Berlin.

The chief objection to all *other media* outside of blood-serum appeared to be that ordinary moulds and bacteria grow better in them than the actinomyces.

In the first generation the fungus did not appear to grow well, but in the second the crop proved successful. Some of the cultures I carried to the fourth generation, which appears to be the limit of prosperous growth in artificial culture for the actinomyces. Beyond the fourth generation, *e. g.*, in the fifth or sixth, I usually failed to obtain a good crop of the fungus. The best crops were obtained in the second and fourth generations, where the fungus appears to reach the perfect size and development, or the stage of tufts. In the third and fourth generations the stage of tufts was less often reached than in the second, and the fungus occurs only in radiating fragments with imperfect conidiæ.

When these imperfect conidiæ were, however, inoculated into an animal they usually developed into perfect tufts.

The fungus, as I observed from many trials, grows best to its full development in blood-serum at the bodily temperature, or at least transforms at this temperature into those radiating clusters which we are accustomed to see in the lesion of actinomycosis.

The growth of each generation took one week, on the average; further than one week the fungi would not grow well, unless transplanted to fresh culture soil.

The fungus in culture appears to the naked eye in successful cultures as a yellow or grayish-yellow film closely resembling lycopodium, which in growth keeps at the surface as long as it is pure and not contaminated. This appearance of the fungus enables one to recognize it promptly by the unaided eye. It does not liquefy the serum. If liquefaction or mould of any other color or form than above stated appear, it shows that the culture is impure.

ACTINOMYCOSIS IN MAN: WITH REPORT OF CASES.

Although about one hundred cases of actinomycosis in man appear to have been recorded abroad, but six authenticated cases are found in American medical literature; these cases are two by Dr. Murphy, of Chicago (*New York Medical Journal*, 1885, vol. xli. pp. 17–19); one by Dr. Schirmer (*Chicago Medical Journal and Examiner*, vol. liii. p. 354); one by Dr. Ochsner (*Ibid.*, vol. liii. pp. 1–3); and two by Dr. Senn, of Milwaukee (*Transactions of the American Surgical Association*, vol. vi., 1888), to be quoted below. Hence, I think the case which came under my observation worthy of record.

Wm. M. H., æt. 32 years; born in England; occupation, miner; mother, four brothers, and three sisters living in good health; father died of senile debility. Patient had ordinary diseases of childhood, besides pleurisy at the age of twenty-eight years. He came to this country at the age of twenty-one years, and continued his occupation steadily as a miner, and he was often subjected to injuries of the head. In 1877 he was severely struck on the right side of the head by a prop. He continued to work in the mines, and while in the Stockton Coal Company's mines, Luzerne county, Pa., he noticed for the first time a swelling at the angle of the right inferior maxillary bone, which would go and come, and, as the patient expressed it, came to stay in the winter of 1882, and soon after reached the size of a chicken's egg, when he became alarmed and began poulticing it; at the same time he had the three upper and three lower back molars of the right side drawn. All six teeth were sound.

Patient states he was advised to keep the gums bleeding and irritated, believing this treatment would reduce the swelling and effect a cure. A growth, however, promptly made its appearance, filling up the cavity made by the extraction of the teeth of the lower and upper jaws. About this time he ran against a projecting drill (an iron rod six feet in length, with a sharp, square end two inches broad), which severely injured the tumor; the wound bled profusely, but healed promptly.

During 1883 the swelling made little progress, and in May, 1884, he came to Philadelphia, and was admitted to the surgical wards of the Pennsylvania Hospital, where, as he states, a tumor was removed. He then returned to work, and had no medical treatment for four years, when he again entered the Pennsylvania Hospital in November, 1888,

having in the meantime sustained some more injuries of the diseased jaw. There is no evidence that a diagnosis of actinomycosis had been made.

Fig. 4.

Case of actinomycosis.

On January 11, 1889, he came under my observation at the German Hospital, when I examined him in conjunction with Drs. John B. Deaver and F. Gross.

Present condition: Patient is a muscular, well-nourished, robust man, of five feet five and a half inches in height, with good complexion, weighing one hundred and fifty pounds, and has a good appetite. The only lesion observed is a swelling on the right side of the head, face, and neck (see Fig. 4, from a photograph), which consists of a hard, infiltrated, uniform mass, extending from the clavicle to the temporal region, involving the anterior half of the temporal muscle, the muscles of the face, and the glands of the neck, and evidently in intimate union with the cranial bones of the region described. The swelling implicates also the cavity of the mouth; the buccal muscles, as well as the lower and upper jaw, are much enlarged from the swelling, which

causes an ankylosis, so that the patient cannot separate his teeth more than a quarter of an inch, and is unable to take food except by forcing it through this narrow space; this, the patient said, has been the case

Fig. 5.

Actinomyces.

for the last two years. While mastication is next to impossible, the power of deglutition appears to be perfect. He uses mainly food in a crushed or liquid state.

The swelling is uniformly firm, except in the temporal region, where there is a fistulous opening; another fistulous opening is at the angle of the jaw, but numerous scars upon the neck indicate that there were a number of fistulous openings which have healed. A third fistulous opening into the mouth corresponds to the third lower molar tooth.

The patient states that there had been a great deal of suppuration from the former fistulas. On the whole, the right side of the head, face, and neck is about one inch thicker than the left side; the pain is slight, somewhat increased by pressure, and is more intense during the night. A large scar, extending from the angle of the mouth to the lower border of the inferior maxilla, indicates a former removal of some of the tumor mass in 1884, at the Pennsylvania Hospital, as the patient states.

The discharge from the fistulous openings is sometimes copious, but at present not more than a few drops at a time; it consists of a purulent, odorless, viscid liquid, of a grayish color, and suspended in it are numerous yellowish granules, distinctly recognizable to the naked eye, being of the size of small poppy seeds. These granules I found under the microscope to be actinomyces, the radiating fungus peculiar to actinomycosis, and I diagnosed the case accordingly.

The fluid in which the fungus is suspended contains numerous compound granule cells, granular material, pus corpuscles, and fragments of fibres in a state of fatty metamorphosis; the fungus presents itself in its perfectly developed or adult form as radiating tufts, round or elongated, measuring $\frac{1}{200}$ to $\frac{1}{50}$ of an inch in diameter; the mycelia are unusually plain and single free mycelia or clusters of two or three, as I have seen in animal actinomycosis, are quite scarce (Fig. 5).

The patient was operated on by Dr. Deaver, assisted by myself, February 9, 1889. An exploratory incision being made from the fistulous opening in the temporal region to within one inch of the angle of the inferior maxilla, and the skin which was involved dissected back, disclosed a very vascular infiltrated mass, fibroid in character, with actinomyces studded through its entirety; also, two cavities one-half inch in diameter, which contained material in a state of fatty metamorphosis which, under the microscope, revealed pus, compound granule cells, blood, oil globules, molecular débris, and the fungus actinomyces.

There was a profuse general oozing of blood over the whole surface of the exposed swelling. A sinus led from the fistulous opening in the temporal region down to the zygoma, which was necrosed, and beneath the same into the pterygo-maxillary region.

A part only of the tumor mass was removed, and the exposed growth was thoroughly curetted and irrigated with 1:1000 sublimate solution; a drainage-tube was inserted, the skin adjusted, and the wound dressed antiseptically.

The six cases of actinomycosis in man recorded in this country previous to this case of mine are as follows:

Case I.—A female, æt. 28 years, suffered severe toothache of left

lower jaw, with subsequent swelling in mouth and throat, the swelling (abscess in left tonsil) was opened, and patient recovered rapidly. About seven months later a swelling, the size of a walnut, appeared on the left side of the neck, which was lanced; the pus contained actinomyces. As the swelling and induration continued to increase, an operation was performed and the growth removed. Patient made a rapid recovery, gaining twenty-six pounds in five weeks, and now (1885) is in good health.—Dr. Murphy, *New York Medical Journal*, 1885, vol. xli. p. 17.

Case II.—Thomas C., æt. 18 years, while in Ireland, had severe toothache; later, a swelling appeared and enlarged with more or less pain; a carious tooth was found, a swelling was punctured, and a few drops of pus escaped which contained actinomyces; the sinus was scraped out, and the parts completely healed. Later, a swelling reappeared in the same place, which was opened, and a drainage-tube inserted, after which Dr. Murphy did not see the case, but heard that the opening healed.—(*Ibid.*)

Case III.—A Pole, æt. 25 years, family history good. When 18 years of age, while chopping wood, received a slight incised wound of cheek. Following this injury both jaws began to swell; the swelling in the left side disappeared, and that on the right side was opened and a fistula followed. In June, 1886, patient passed into Dr. Schirmer's hands. The exudate from the fistulous opening was examined by Professor Fenger and Dr. Schirmer, and actinomyces was found. Patient lost his appetite, became emaciated, followed by severe cough, and the expectoration contained actinomyces.—Dr. Schirmer, *Chicago Medical Journal and Examiner*, vol. lii. p. 354.

Case IV.—A man, æt. 56 years, whose occupation was stock-raising and dealing, was much exposed to draughts and cold. He suffered pain in the left antrum of Highmore, and although he had six teeth drawn which were sound, no relief followed. For six months patient suffered excruciating pains in left antrum and both eyes. Early in 1878 there was a spontaneous opening of the abscess into the pharynx, from time to time evacuating considerable pus and blood; some of the discharge entered the larynx at night, giving rise to severe cough. In 1880 was operated upon, curetting and irrigation of the parts resorted to; irrigation was kept up two years, patient suffering continuous pain. In 1885–6 severe cough, with dulness over lungs, roughened respiratory sounds and mucous râles, followed by great loss of weight. Microscopic examination of the sputum revealed actinomyces.—Dr.

Ochsner, *Journal of American Medical Association*, 1886, pp. 608–610; *Chicago Medical Journal and Examiner*, vol. liii. pp. 1–3.

Case V.—A man, æt. 30 years, German by birth, a soda-water manufacturer. His business required him to make frequent trips into the country. During the winter of 1886 suffered from an ordinary cold; the right side of lower jaw was swollen and painful; one of the molar teeth showed evidence of decay, and was extracted. The pain and swelling did not improve, after which all the molars of that side were extracted. After this a fungous mass commenced to appear. Actinomyces were found in the pus from the small abscess cavities and surface of the fungous granulations.

Patient was operated upon, the affected parts removed, and the exposed bone surface thoroughly cauterized. The deep portion of wound was dusted with iodoform and filled with iodoform gauze, while the external wound was sutured, and healed by primary union, and the cavity in the mouth closed slowly by granulation.

Patient's general health improved rapidly until six weeks after the operation, when the neck below the scar became swollen, followed in a short time by formation of abscesses. Efficient drainage was established, but suppuration continued. The patient became extremely emaciated. Four months after first operation he died. Dr. Senn believes that recurrence of the disease was due to imperfect removal of infected tissue in the posterior and lower portion of the pharynx.—Dr. Senn, *Transactions of the American Surgical Association*, vol. vi. 1888.

Case VI.—This case came under the care of Dr. Senn in the summer of 1887. Patient was a young man, employed on a farm. Five months before his admittance to the Milwaukee Hospital he had a number of teeth extracted, believing that the teeth, some of which were decayed, were the cause of the pain and swelling in that region. His physician diagnosticated sarcoma of the upper jaw, and sent the case to Dr. Senn for operation. The swelling involved the right side of the face, extending from the zygomatic arch to near the lower border of the lower jaw, involving the deep tissues, and being connected with the alveolar processes of the posterior portion of the upper jaw. The swelling was firm, and without well-defined margins. No evidences of suppuration. All infected tissue was thoroughly excised, the jaw-bone was scraped and cauterized. The entire thickness of the cheek, with the exception of skin and superficial facia, appeared to be transformed into granulation tissue, in which actinomyces were found. The wound healed, and the patient has remained in perfect health since the time of operation.—(*Ibid.*)

The More Important European Cases of Actinomycosis in Man.—Langenbeck observed unquestionably a case of actinomycosis in man in 1845. A patient, supposed to suffer from tubercular disease, was admitted to the surgical clinic of von Langenbeck, then at Kiel. The patient had caries of the dorsal and lumbar vertebræ with some fistulæ opening in the covering skin, from which an ichorous pus containing yellow granules of the size of a poppy-seed exuded; these granules were regarded as cheesy, tubercular masses. But microscopical examination made by von Langenbeck, and recorded at the time of examination, showed these masses to consist of fungi, the nature of which was not determined at the time, but which, as it is now evident from the original drawings made at the time, was the actinomyces.

The description given leaves no doubt about the identity. The clinical features of the case, as well as the subsequent autopsy record, confirm such presumption. The vertebræ and the surrounding soft parts presented new formations with purulent masses filled with yellow granules and other lesions fully corresponding to some of the cases described subsequently by recent observers of actinomycosis. The description of the above case of Langenbeck's was resurrected by Israel from the autopsy book of the Friedrich's Hospital at Kiel.

J. Israel (*Virchow's Archiv*, vol. lxxiv.) in 1878, or thirty-three years later, next described four cases of a new mycosis in man, identical with the case related by Langenbeck. These also were, evidently, genuine cases of actinomycosis, judging from the excellent description of the lesion and of the fungus given by Israel, and the identity with the disease in animals as described first by Bollinger in 1877. But Israel, at the time of the publication of his cases, was not aware of this fact, and hence he, and not Ponfick, should be credited with the discovery of actinomycosis in man, even if he did not term the disease by its proper name. Israel's cases are so exhaustively described in *Virchow's Archives*, and with such precision, that I cannot refrain here from giving the details.

Analysis of the cases referred to by Israel shows that they represent:

1. A case of a pleural affection in a woman æt. 39 years, resembling tuberculosis, but characterized by caries of the ribs and spine, from which fistular openings led externally through the skin. The pus exuding contained the unquestionable actinomyces fungus, as may be inferred from his excellent description and drawings. This case of Israel's terminated fatally by "chronic pyæmia," as he says. All the lesions found post-mortem correspond to actinomycosis.

2. A case of a man æt. 36 years. This case was one of inflammation

tumefactions of the alveolar processes of the lower jaw, with deep-seated abscesses, fistular openings and exudations containing actinomyces granules. Recovery.

3. A case of a girl, æt. 9 years, suffering from sub-periosteal abscesses at the margin of the lower jaw; coincident with caries of the third molar; from fistular opening exuded pus containing the yellow granules. Recovery.

4. A case in all respects analogous to Case 1, and terminating also fatally.

The name by which Israel designated the fungus found was in accordance with the suggestion of Ferdinand Cohn—*Streptothrix Försteri.*

Ponfick (*Die Actinomykose des menschen*, Berlin, 1882) describes his five cases of actinomycosis with full details. The first of these, and the one which he claims as representing the discovery of actinomycosis in man, or at least the first observation of this disease made knowingly in man, is published in the *Breslauer Aertzlicher Zeitschrift*, 1879.

This case of Ponfick's was that of a powerfully built man, æt. 45 years, who had suffered from the sequelæ of pleurisy on the left side for a year and eight months. After death there was found an extensive prevertebral phlegmonous inflammation in the posterior mediastinum, with a a parapleuritic abscess cavity extending both to the right and left, at the level of the seventh, eighth, and ninth intercostal spaces; with this cavity there communicated a complex system of sinuses, extending through the substance of the longissimus dorsi, the scapular muscles, and the subcutaneous tissues of the whole back. The sulphur-yellow fungous bodies were found upon or between the granulations of these sinuses, and in their substance as well as in their sero-purulent discharge; they were also found in a cavity of the size of a cherry, which occupied the centre of a hepatized area of the left lung (lower lobe), as well as in the exudation that filled some of the neighboring alveoli.

The second case was that of a woman, æt. 61 years, admitted with an abscess of the lower part of the abdominal wall; she subsequently developed another abscess of the left iliac fossa, without recurring symptoms of peritonitis, and died from exhaustion. After death caries (with prevertebral collection of pus) of the three lower lumbar and first sacral vertebræ, abscesses in both iliac fossæ, and perityphlitic adhesions were found. The yellow fungous bodies were discovered in the pus of the prevertebral abscess.

The third case was that of a woman, æt. 45 years, who had suffered an injury of the right thumb three years before, with swelling of the arm, which did not subside, but extended to the neck and back, and was

accompanied by progressive weakness. The necropsy revealed extensive sinuses on the left side of the neck and in the prevertebral tissue, a knob-like excrescence of new growth extending into the lumen of the internal jugular vein, a tumor of the size of an apple, growing into the right auricle and ventricle, with corresponding whitish centres in the ventricular substance and gelatinous nodules in the spleen and in the occipital lobe of the right cerebral hemisphere. In this remarkable case the fungous bodies were found in the sinuses of the neck, in the substance of the sarcoma-like growth of the jugular vein, in the tumor of the right auricle and ventricle, and elsewhere.

In the fourth case the illness began fourteen months before death, following the extraction of an upper molar tooth; it consisted of swelling in the region of the right maxillary joint, tumefaction of the face, and subsequently of the neck; successive outbreaks of abscesses and sinuses in these regions. Death occurred from extreme exhaustion. The yellow fungous bodies were frequently obtained in this case from the sinuses during life. The record of the case is too elaborate to be given, even in outline; but it may be mentioned that there was, besides the extensive sinuses and granulation centres of the face and neck, a prevertebral abscess extending from the basilar process of the occiput to the fourth dorsal vertebra, with osteophysic growth from all the bones, together with caries of both occipito-atlantal joints and of the right atlanto-axial.

In the fifth case, a boy, the first indication of illness was a year before death, when he had symptoms of pleurisy; nine months later there was a new and much more severe affection of the same side, with general dropsy, progressing swelling in the lower part of the back, and evacuation of pus from a cavity opposite the eleventh left rib.

Post-mortem.—A large prevertebral cavity was found on the left side, partly retropleural at the level of the eighth, ninth, and tenth ribs, and partly retroperitoneal at the level of the last two ribs and the left kidney; also, several perforations of the diaphragm. There were numerous centres of actinomycosis in the muscles of the back, in the intercostals, and in the left psoas muscle; also, in the muscular substance of the left ventricle and in the upper end of the spleen.

Four cases of actinomycosis, with extensive tumefaction and deep-seated abscesses, with lesions having started, as usual in man, from the soft parts and subsequently proceeding to the bones, are recorded by Rosenbach ("zur kenntniss der strahlenpilzer krankungen beim menschen"), *Centralblatt für Chirurgie*, 1880, p. 225. Rosenbach claims that actinomycosis is a very common affection among the country people in

the neighborhood of Göttingen, but that the affection is usually local and quite amenable to treatment. Further, he stated that the disease commences usually in the neighborhood of the jaws, and appeared to be dependent upon carious teeth, involving the dental alveoli.

The same suggestion was also made by Ponfick and Israel. In mankind the tendency of the disease is to formation of abscesses and suppurations; in bovines there is more of a tendency to tumor formation. Not having access to Rosenbach's original paper, I cannot give details; but from the brief quotation of Ponfick and Fleming the cases were fully analogous to the cases before described, and the actinomyces fungus was observed in all lesions.

Five cases of actinomycosis affecting the peritoneum and abdominal organs are reported from Vienna by Dr. Adolf Zemann,[1] assistant at the Pathological Institute there. The characteristic fungous colonies were found in all cases, both in the purulent contents of the abscesses and in the tissues of the affected parts. The number of previously reported cases is small. Ponfick collected sixteen in all of his own and others, and these were published in his monograph on the subject, which appeared in 1882. These cases Ponfick divided into two groups—those which recovered and those which were fatal. The former group consisted of cases where the malady made its appearance on the jaws, preferably on the lower jaw, and spread downward along the neck to the clavicle.

The second group of cases comprised those in which the process announced itself by pains in the limbs, or more generally along the spinal column and neighboring parts of the back, succeeded by pneumonia or pleuro-pneumonia of the left lower lobe. In a few cases these pains precede prevertebral gathering which becomes a psoas abscess.

The five cases now reported seem therefore to be not only a considerable addition to the existing observations of a but little known pathological process, but also to enlarge the clinical description of the disease. Some of these cases ran their course with fever and others without. Zemann concludes from his study of them that the important point in the diagnosis of actinomycosis is the discovery of the pathognomonic fungus. Apart from such identification the disease can neither be diagnosticated from the part affected nor from the course which it runs, since the process may be febrile and acute, or almost without fever and chronic; in which latter case it manifests itself as an induration, as a chronic inflammation of serous membrane, as an inflammation in the

[1] Wiener med. Jahrbuch, 1883, p. 477.

bones or periosteum, or as an inflammation attacking the internal organs. All of which is demonstrated by the varied points from which the fungus gains access to the body, and by almost innumerable complications attendant on its presence.

Zemann's cases certainly tend to show that by no means so simple a picture as that given in Ponfick's monograph will suffice to represent actinomycosis.

There are a few cases of actinomycosis said to be described by Kracke, Esmack, Weigert, and Partsch (*Breslauer Aerztliche Zeitschrift*, p. 78, 1881), but I had no access to the original papers, and hence can give no details.

All the above cases of actinomycosis, as seen in man, have been described in Germany and by German observers. The only case described elsewhere (up to 1884) is that of W. R. Treves, of London, surgeon to the National Hospital for Scrofula. The case is as follows:

"P. C., æt. 45 years, a brickburner, was admitted on August 17, 1883, into the Royal Sea-bathing Infirmary under my care, supposed to be suffering from a scrofulous affection of the glands of the neck. He is a powerfully built, muscular man, with a good family history. His illness began sixteen months before admission with inflammation about the neck and angle of the jaw, which, however, only kept him from work some two or three days. He has lived well and been in a comfortable position for his class in life. He has never had cattle to look after. A lump near the angle of the jaw, followed by inflammation, was incised. Subsequently other swellings formed. When first seen he was in fair general health and well nourished. Over the angle of the jaw and in the posterior triangle of the neck were three ulcerated and fungating surfaces, those near the angle of the jaw being about one inch, and that in the posterior triangle about two inches in diameter. There were tumors over the collar bone, the second rib, and the fourth costal cartilage near the sternum, each of which was in a direct line, and had followed in regular order the one described as existing in the posterior triangle. These tumors resemble each other in appearance; they are smooth and evenly formed, and are in shape as nearly as possible half a sphere; the upper one is two inches in diameter, the lower one an inch, and the middle one intermediate in size; they have an elastic, semi-fluctuating feel; the skin over the upper one is thin, red, and evidently about to open; the skin of the middle one is also discolored; that of the lowest is normal. To the right and left of these tumors are two nodules about the size of a marble, apparently the same thing in process of formation. The discharge was thin and serous and contained minute

yellowish masses and disintegrated tissue, and had a peculiarly offensive and sour smell. The patient declined operative interference. He remained in the hospital till December 7th, the progress of the case being gradual loss of flesh, formation of lumps on the other side of the neck and in the axilla. The appearance of the tumors resembled nothing I had ever seen before. The case was certainly not scrofulous; nor was it like any new growth with which I was familiar. I arrived at the conclusion that it was an example of the disease known as actinomycosis. This diagnosis was confirmed by the discovery under the microscope of bodies which I believed to resemble the fungus described as peculiar to this disease. So far as I can ascertain, this is the first case of actinomycosis described in this country."

Since the publication of Dr. Treves's article numerous cases of actinomycosis in man have appeared in the literature—in all over one hundred cases—due reference to which the reader will find in the bibliography to this paper:

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